THE UNIVERSITY OF ALBERTA

EFFECTS OF TASKS OF VARYING COMPLEXITY ON ELECTRODERMAL AND HEART RATE MEASURES IN SCHIZOPHRENICS

by

© ORESTES FEDORA

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF PSYCHOLOGY

EDMONTON, ALBERTA

FALL, 1976

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to The Faculty of Graduate Studies and Research, for acceptance, a thesis entitled, "Effects of Tasks of Varying Complexity on Electrodermal and Heart Rate Measures in Schizophrenics" submitted by Orestes Fedora in partial fulfilment of the requirements for the Degree of Doctor of Philosophy in Psychology.

Ror E. Walley
Supervisor

Lorne T. Goudall
Co-supervisor

John Ursched

Joseph R. Royce

Merle M. Bowsma

Roland D. Hare
External Examiner

DATE September 24, 1976

ABSTRACT

The view that certain psychopathological states, such as schizophrenia, are associated with brain dysfunction is presented together with evidence supporting such a position in the introduction. Specifically, the relationship between one psychopathological state, schizophrenia, and changes in psychophysiological response measures is reviewed. Several studies (Ax and Bamford, 1971; Bernstein, 1970; Gruzelier, 1973; Gruzelier and Venables, 1971, 1972, 1973, 1974; Mednick, 1970; Mednick and Schuslinger, 1968; Stern, Surphlis and Koff, 1965; and Venables, 1972) have demonstrated that psychopathological states, including schizophrenia, differ with respect to psychophysiological responses recorded in a variety of task situations compared with normal controls.

In the present study an attempt was made to determine the differences in psychophysiological responses between two schizophrenic groups pre-selected on the basis of a criterion measure: electrodermal reactivity during the performance of several cognitive tasks. Schizophrenics showing electrodermal responses to the cognitive tasks were designated responders; schizophrenics showing no electrodermal reactivity during the performance of these tasks were termed non-responders. A third group was comprised of normal controls.

A number of task situations were employed which varied in complexity with respect to physical, emotional (stress) and cognitive demands. Thus, subjects were tested in situations of low physical and cognitive demand (low stress situations such as repeated pre-

senations of tones, or discrimination of two very different tones: 1000 and 2000 Hz), situations with a larger physical demand (such as taking deep breaths or coughing hard upon command), situations with both physical and emotional demands (such as those involving shock presentation and startle stimuli) and situations involving considerable cognitive demand (such as spelling backwards, arithmetic, and mirror tracing problems).

Respiration responses, heart beat intervals and bilateral skin conductance responses were recorded from all subjects during the performance of the tasks. In addition, a WAIS and modified Halstead-Reitan Neuropsychological test battery were administered to most of the subjects in the study.

The two schizophrenic groups differed from the normal controls and from each other with respect to several of the psychophysiological measures as well as on the psychological measures. The responders and nonresponders had higher respiration rhythms, higher heart rates, and lower SC levels compared with controls although the latter two effects were confounded with medication level. The responders had more spontaneous fluctuations and the nonresponders had less spontaneous fluctuations compared with controls during the performance of the tones habituation task. The nonresponders has significantly smaller amplitudes of SCRs on all tasks except the least stressful task (repeated presentations of tone) compared with responders and controls. Nonresponders had slower latencies of SCRs compared with responders and controls. The responders had shorter ascent times for SCRs on several tasks compared with controls while nonresponders

and controls did not differ with respect to ascent time of SCRs. The nonresponders did not differ from the controls on the recovery time of SCR measure, although a trend toward longer recovery times was observed for the nonresponder group. The responders also did not differ from the controls on the recovery time measure, but a trend toward shorter recovery times during the performance of the tones habituation task was observed for the responder group. Finally, the nonresponders were found to be more chronically ill and to show significantly more impairment on the psychological variables: the WAIS and the neuropsychological test battery.

Similarities and differences between the present study and related work is discussed and the data interpreted to be consistent with several behaviorially-based theories of schizophrenia, including that of Broen (1968). The two groups of schizophrenics are seen to represent distinct populations with varying forms or degrees of brain dysfunction such as has been described by Gruzelier and Venables (1972, 1973) and by Kornetsky and Mirsky (1966). The results are discussed in terms of the extent to which they may be accounted for by concepts such as that of Pavlovian protective inhibition.

ACKNOWLEDGMENTS

A number of people have contributed much to this thesis. I wish to thank Dr. Roc Walley, Chairman of the thesis committee, for his academic guidance, continued support, numerous suggestions and critical thinking, as well as for his considerable involvement in the thesis from the beginning to the end of the project without which it would not have been possible. I would like to thank two other members of the thesis committee, Dr. Joseph Royce and Dr. Robert Bourassa, for their time and their willingness to participate in a difficult and complex area of research not particularly related to their own research interests.

I wish to express sincere thanks to Dr. Robert Hare, Associate Professor of Psychology, University of British Columbia, for his careful and considered evaluation of the thesis and for taking seriously his job as "outside member" of the committee.

Others have also contributed in ways that made this sort of thesis possible: Dr. Pierre Flor-Henry and Dr. Brian Howarth, provided the double blind differential diagnosis of schizophrenia in the experimental research groups; they generously contributed valuable time in examining and sometimes re-examining patients at Alberta Hospital, Edmonton; they gave me, as well, insights into the psychiatric views of psychopathological groups. I thank Dr. Cornish, Hospital Director, for permission granted to use the facilities at Alberta Hospital and to test patients for research purposes.

My appreciation and special thanks go to Dr. Lorne Yeudall,

Head of the Department of Neuropsychological Research at AHE, Edmonton, whose constant support, warm encouragement and unflagging interest in all aspects of the research sustained me throughout the more frustrating and difficult stages of the project and I want to acknowledge particularly his contribution as a co-chairman of the thesis and the many hours he spent with me in the role of academic advisor, critic, and friendly supporter.

I thank John Lind, Douglas Wardell and Terry Gee for their aid in analyzing the data and their most helpful guidance in understanding the statistical aspects of the analysis. My sincere thanks also go to Beth Utendale for her corrections of spelling and grammatical errors and typing of the nearly illegible rough drafts of the thesis chapters. I thank Bill Stefanyk for his patience, ability, and persistence throughout many hours of neuropsychological testing of often uncooperative and difficult subjects.

Natalie Daviduk and Marilyn Greig I thank for their preparation of the final draft of the thesis for submission to Graduate Studies with speed and accuracy.

Finally, I wish to thank my wife, Shawn, for helping me to express my ideas more clearly in the writing of the thesis, for providing a "sounding board" for my own critical thinking, and finally, for her considerable personal support throughout my days as a graduate student.

TABLE OF CONTENTS

	PAGE
Abstract	iv
Acknowledgments	vii
Table of Contents	ix
List of Tables	xi
List of Figures	xii
List of Appendices	xv
Introduction	1
Cerebral lateralization of psychological function	2
Lateralized brain dysfunction and psychopathology	3
Human brain damage: Effects on psychophysiological response measures	6
Psychopathology: Effects on Psychophysiological response measures	9
Purpose of study	17
Method	19
Subjects	19
Equipment	22
Experimental procedures	25
Response definition and data quantification	30
Psychological assessment of subjects	32
Results	34
Experimental group differences	34
Skin conductance response frequencies	35
Response amplitude	44

Response latency 48
Response ascent or rise time 52
Response recovery time 55
Skin conductance levels 64
Heart rate response 68
Respiration rate response 71
Neuropsychological data 72
Discussion 75
Significant differences: Group attributes or statistical Artifacts 75
Similarities and differences between the present and other studies 79
General discussion 85
Footnotes 100
References 102
Appendices 115
A Summary of Analyses of electrodermal amplitudes 115
B Summary of analyses of electrodermal latency 129
C Summary of Ascent time and ratio analyses 135
D Summary of SCR recovery time and ratio analyses 142
E Bilateral skin conductance level differences 149
F Summary of WAIS and Neuropsychological variables ... 153
G Medication effects in schizophrenics 157
H Univariate versus Multivariate Statistics 158

LIST OF TABLES

TABLE		PAGE
1	Mean ages of the two schizophrenic groups and control group, mean current length of hospitalization, number of previous known hospitalizations and mean length of total hospitalization for both schizophrenic groups	23
2	Type of and daily drug doses in mg. An equivalent table for the sedative effects of major tranquilizers.	24
3	Spearman rank order correlations of drug dosages with electrodermal measures in the pooled schizophrenic population	36
4	Mann-Whitney U values of both hands for the percentages of possible responses during the first 15 trials and the entire 18 trials of task two for all three groups	38
5	Mann-Whitney U values of both hands for the percentages of possible responses during the signal and neutral tones of task three for all three groups	41
6	Number of spontaneous fluctuations for both hands during the habituation/dishabituation task for all three groups..	43
7	Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean SCR amplitude during other tasks	49
8	Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean ascent times and mean ascent ratios during several tasks	56

LIST OF FIGURES

FIGURE		PAGE
1	Percentages of subjects responding for the three groups during habituation and dishabituation of task two	37
2	Percentage of trials with SCRs occurring during the signal and neutral tones of task three for three groups	40
3	Mean amplitudes of SCRs for the R and L hands of the three groups during eleven situations	45
4	Mean amplitude of SCRs, with zero amplitude SCRs excluded, for three groups during four events	47
5	Mean latencies of SCRs for the R and L hands of the three groups during seven situations	50
6	Mean ascent times and ascent ratios of the R and L hands for the three groups during 13 situations	54
7a	Mean one-half amplitude recovery times of the R and L hands for the three groups during 14 situations	59
7b	Mean one-half amplitude recovery ratios of the R and L hands for the three groups during 14 situations.	61
8	Mean R and L hand SC levels for the three groups determined just prior to the SCRs for 7 situations	65
9	Mean SCL change scores during cognitive tasks four, arithmetic, and five, spelling backwards, for the three groups	67

9 Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean recovery times and mean recovery ratios during several tasks 63

10 Basal mean heart rate before and after habituation of task two 69

11 Mean inspirations per minute for each group and Mann-Whitney U comparisons between groups 73

1	Mean amplitudes of SCRs for the R and L hands of the three groups during habituation and dishabituation of task two	121
11	The absolute SCL differences between R and L hands and the index scores of absolute SCL differences for the three groups during six situations	152

INTRODUCTION

The current view that certain psychopathological states, such as schizophrenia, are associated with brain dysfunction has received attention from a number of independent but related sciences. These relatively different approaches include research in biological psychiatry, clinical psychology, neuropsychology, and psychophysiology.

There have been several reports in the research literature which have consistently noted the similarity of certain psychiatric disorders associated with temporal lobe epilepsy, a condition known to be associated with brain dysfunction, to certain other psychopathologies, including schizophrenia. Secondly, there is a considerable amount of research using EEG techniques and neuropsychological methods to investigate lateralized brain dysfunction and its relationship with the various psychopathologies. Flor-Henry (1972) using these techniques has published a conceptualization of schizophrenia in terms of lateralized brain dysfunction in the anterior cortical and limbic systems of the brain. Thirdly, clinical studies of the psychophysiological correlates of human brain damage by Soviet psychologists, such as Luria and Homskaya (1966, 1970) have contributed to the understanding of the nature of psychophysiological deficits associated with relatively discrete areas of brain damage. Evidence for the lateralization of psychophysiological functions is also available, though to a limited degree (Holloway & Parsons, 1969; Parsons, 1970).

In the course of their development, these relatively independent areas of research have often overlapped, drawn on each other and even

coalesced. For example, Gruzelier and Venables (1971, 1972) using techniques employed earlier by Soviet psychologists (Luria & Homskaya, 1966, 1970) to assess changes in the psychophysiological concomitants of the orienting response (OR), attempted to test the hypothesis of Flor-Henry (1969) that certain psychiatric disorders may be characterized in terms of lateralized brain dysfunction. Neuropsychological methods of assessment have been employed to provide evidence that schizophrenia and several other psychopathologies are characterized by lateralized cerebral dysfunction (Yeudall, 1976).

In spite of the considerable degree of overlap among the scientific disciplines involved in such research an attempt has been made throughout this chapter to categorize the various areas of investigation for the purpose of seeing more clearly the nature of their empirical contributions to the conceptualization of schizophrenia in terms of brain dysfunction. Since any discussion of lateralized brain dysfunction presupposes a familiarity with evidence for such a process a general statement of the findings is presented below.

Cerebral Lateralization of Psychological Function

That the two hemispheres of the brain are differentially organized in relation to various psychological functions is a viewpoint currently gaining general recognition. There is a growing body of evidence that such is the case (for example, see Benton, 1969; Diamond, 1972; Flor-Henry, Koles, Bo-Lassen & Yeudall, 1975; Gazzaniga, 1970; Gazzaniga, Bogen & Sperry, 1965; Milner & Taylor, 1972; Mountcastle, 1962; Penfield, 1971; Reitan & Davison, 1974; Zangwill, 1963).

The results of such research into the lateralization of psychological functions are consistent with the position that both hemispheres appear

to be involved in the processing and organization of both verbal and nonverbal information, with the dominant hemisphere relatively more involved in the processing of symbolic language and propositional speech and the nondominant hemisphere relatively more involved in the processing and organization of visual, spatial, tactual, and musical information.

Lateralized Brain Dysfunction and Psychopathology

The similarity of psychopathologies to certain forms of brain dysfunction such as organic brain damage and epilepsy has been noted from time to time in the literature. Bradley (1951) concluded that several primary symptoms of epilepsy such as erratic mood, hypermobility, irritability, short and vacillating attention, and a rather selective difficulty in mathematics have a striking similarity to the early signs of schizophrenia. Slater, Beard, and Glithero (1963) have proposed that temporal lobe epilepsy might serve as a mockup of certain forms of schizophrenia. White (1974) also conceptualizes schizophrenia in certain forms and temporal lobe epilepsy as being on a neuroanatomical continuum.

Flor-Henry (1969) has reported a significant correlation between psychiatric diagnosis and epilepsy but with reference only to the side of the brain affected by temporal lobe epilepsy. Schizophrenia was found to be more frequently associated with left temporal lobe epilepsy whereas manic-depressive illness was more frequently associated with right temporal lobe epilepsy. In a series of review papers Flor-Henry (1972, 1973, 1974) further elaborated his hypothesis that epilepsy and a number of psychopathologies are characterized by lateralized brain dysfunction.

Specifically, Flor-Henry (1969, 1972) hypothesized that schizophrenia, psychopathy, hysteria, and the periodic-affective disorders are characterized by lateralized cerebral dysfunction of the anterior cortical and limbic regions of the cerebral hemispheres. Furthermore (Flor-Henry, 1973, 1974), it was suggested that the periodic-affective disorders are characterized by a dysfunction predominantly of the nondominant temporal-limbic system with bilateral involvement of the orbital-frontal extension of the limbic system. Psychopathy, hysteria, and schizophrenia, on the other hand, were conceptualized as having "perturbations" predominantly of the temporal-frontal limbic systems of the dominant cerebral hemisphere, a view without specific precedence in the literature on psychopathology.

Flor-Henry, Koles, Bo-Lassen, and Yeudall (1975) using power spectral analysis (Fast Fourier Transform) analyzed the EEG records of schizophrenics and manic-depressive patients who were medication free, as well as the EEG records of normal controls in a variety of psychological task situations. The authors confirmed the results of Galin and Ornstein (1972) who found that the changes in the right-left energy ratios of the two sides of the brain during verbal and spatial tasks were related to the hemispheres involved in these tasks. That is, relatively greater alpha suppression occurred in the dominant hemisphere, compared with the nondominant hemisphere, when the subject was engaged in verbal tasks, and the reverse occurred when the subject was engaged in spatial tasks. The authors also reported that schizophrenics were found to have a greater proportion of 20-30 Hz activity in their left as opposed to their right temporal regions, and compared with controls, they had significantly more 20-30 Hz activity in their left temporal regions.

The manic-depressive patients had a greater proportion of 20-30 Hz activity in both temporal lobes in comparison with normal controls. In contrast to the schizophrenic group, the manic-depressive group had significantly more fast activity in the right temporal regions.

Neuropsychological assessment techniques (Filskov & Goldstein, 1974) have proved useful in providing further evidence for the association of lateralized brain dysfunction and psychiatric disorders. Yeudall and Flor-Henry (1975) found that the psychiatric diagnosis of depression versus aggressive psychopathy in patients referred for neuropsychological assessment correlated .82 (fourfold point correlation or ϕ coefficient, see McNemar, 1957) with right versus left brain dysfunction as assessed by a test battery consisting of 27 neuropsychological variables and the Wechsler Adult Intelligence Scale (WAIS). That is, depression was associated with right hemisphere dysfunction and aggressive psychopathy was associated with left hemisphere dysfunction. A discriminant function analysis of the neuropsychological and WAIS variables for the psychopaths (N = 25), depressives (N = 25), and a normal control group (N = 25) yielded correct classifications of 84, 88, and 96%, respectively. When patients with documented organic brain damage (psychopaths, N = 3; depressed, N = 9) were excluded from the sample, a discriminant function analysis yielded 95.45, 93.75, and 100.00% correct classifications for the psychopaths, depressives, and normals, respectively.

In a double-blind study (Flor-Henry, Yeudall, Stefanyk, & Howarth, 1975) 115 consecutive admissions of schizophrenic and periodic-affective disorder patients were administered a neuropsychological test battery. Of a total of 53 schizophrenics, 45 showed more left than right hemisphere dysfunction, 2 had bilateral dysfunction and 3 had no

neuropsychological dysfunction. On the other hand, of the 49 patients with a diagnosis of periodic-affective disorder, 45 had right greater than left hemisphere dysfunction, 3 had bilateral dysfunction, and one had no neuropsychological dysfunction. The χ^2 with a Yates correction was equal to 78.02 ($p < .001$). A fourfold point correlation of right versus left hemisphere deficits and periodic-affective disorders versus schizophrenia was .94. A stepwise discriminant function analysis of the neuropsychological test variables yielded 85 and 94% correct classification of the schizophrenic and periodic-affective disorders, respectively.

To briefly summarize what has been presented thus far, many authors have suggested a relationship between brain dysfunction and psychopathologies in studies encompassing divergent and multidisciplinary techniques. Although none of the authors of the studies reviewed suggest that organic brain damage or epileptic states are comparable to psychopathological states, they do suggest by implication that localized dysfunction of the brain may be responsible for some of the symptomatology that is demonstrated by the patients in these studies.

Since psychophysiological abnormalities have been found to be associated with brain damage, the possibility arises that similar psychophysiological impairments may be associated with certain psychiatric or psychopathological states, such as schizophrenia. Evidence for psychophysiological deficits and their relationship with unilateral and bilateral brain damage will be described briefly in the next section.

Human Brain Damage: Effects on Psychophysiological Response Measures

Investigations of central nervous system (CNS) dysfunction and

electrodermal activity in man have been limited in number and scope. However, it is known that patients with brain injuries frequently have multiple symptoms relating to vasomotor instability of the body (Guttman & List, 1928; Hoff, Kell, & Carroll, 1963; Ingram, 1960; Sourek, 1965). Unilateral cortical lesions alter the amplitude of skin potential responses and produce increases in sweating on the side of the body contralateral to the lesion (Guttman & List, 1928).

A considerable portion of the literature on autonomic correlates of brain damage and orienting behavior has been published by Soviet psychologists (Anonkhin, 1935, 1955; Bernstein, 1935, 1947, 1961; Luria, 1966b; Luria & Homskaya, 1966, 1970; Sokolov, 1963). In normal controls, novel signals produced psychophysiological responding which habituated with repeated presentation of the signals. Thus tones or lights would initially produce a whole series of changes (depression of the alpha rhythm, appearance of electrodermal reactivity, vasomotor constriction of the limbs and vasomotor dilation of the forehead) which disappeared with repeated presentation of the signals. Instructions to normal controls, which added signal value to the stimuli (i.e., "estimate the frequency of the tone...give the bulb a short squeeze to a long tone and a long squeeze to a short tone"), resulted in a reappearance of the psychophysiological changes which then persisted for many trials and became so dominant to a particular stimulus that powerful irrelevant stimuli ceased to evoke any appreciable orienting reaction. This stabilized reappearance of autonomic activity is referred to as a "regularization of autonomic activity" by Luria and Homskaya (1966). In patients with massive damage outside the frontal lobes, autonomic reactions to novel stimuli were either absent, present but diffuse and

slow to extinguish, or were paradoxical in character (vasomotor dilation, rather than constriction, of the limb). Furthermore, unlike the control subjects, these patients with massive lesions outside the frontal regions did not demonstrate the appropriate "regularization" of autonomic reactivity with the addition of signal value to their stimuli. However, when these subjects were instructed to verbalize outloud the instructions during each stimulus (i.e., press "strong" with short duration stimulus) the behavioral performance improved and a "regularization" of the autonomic activity was observed. Patients with massive frontal lesions differed from patients with lesions outside the frontal regions in that a dissociation occurred between the frontal patients' behavior and the instructions the patient was verbalizing outloud such that the performance did not improve and the autonomic "regularization" did not occur. In the case of less severe frontal damage, autonomic "regularization" occurred whenever the patient temporarily performed correctly the same task he was verbalizing. Luria and Homskaya (1966) concluded that the degree of disruption to the psychophysiological components accompanying an OR during a habituation task did not appear to be related to either the site or the degree of brain damage, although the regularizing effect of verbal instructions, repeated outloud by the patients, were related to both site and degree of brain damage.

A considerable number of Western studies have been published presenting evidence that cortical and limbic damage is associated with changes in the autonomic aspects of the OR. Holloway and Parsons (1971) reported the expected habituation of skin conductance (SC), heart rate (HR), and alpha blocking components of the OR to an auditory stimulus for the control group. Brain damaged subjects showed evidence of

habituation only for the SC measure. The major findings of the Holloway and Parsons (1971) study were comparable to those reported earlier by Davidoff and McDonald (1964). There was no evidence in either of these two studies for overall group differences in response magnitude. However, in the Holloway and Parsons (1971) study brain damaged subjects had lower initial response magnitudes for the alpha blocking measure, another finding consistent with earlier reports (Blum, 1957; Wells, 1962).

Tonic levels of electrodermal activity have been reported to be higher in cortically brain damaged patients compared with hospitalized controls (Parsons & Chandler, 1969). Unilaterally lesioned patients have higher levels of electrodermal activity contralateral to the side of the brain damage (Holloway & Parsons, 1969).

Since some researchers have reported unilateral increases of skin conductance levels (SCL) on the side contralateral to site of brain damage (Holloway & Parsons, 1969), and others (Luria & Homskaya, 1966, 1970) have reported a lack of psychophysiological responses including electrodermal and vasomotor activity in the brain damaged, it appears that brain damage may result in either increases of SCLs and concomitant increases in skin conductance responses (SCR), or decreases of SCLs and the absence of electrodermal activity. Clinical observations by this author of known brain damaged patients at Alberta Hospital, Edmonton (AHE) verify both types of effects.

Psychopathology: Effects on Psychophysiological Response Measures

Although the attempt to relate bodily processes to psychological states has a long history, no theoretical formulation to date has been broad enough to account more than selectively for a rather extensive

literature. Nor are they precise enough to martial evidence which could reduce the variety of opposing notions. This is so partly because the two domains of psychopathology and psychophysiology are known for their complexity and also because of difficulties in response measurement, lack of reliability in certain parameters and problems in instrumentation. Too few research studies in this area have been themselves products of a systematic theoretical formulation. Examining the reviews of physiological concepts of psychopathology (see, for example, Alexander, 1972) one is left with the impression that there are as many psychophysiological concepts of psychopathology as there are psychological concepts in the field of psychology.

A frequently employed concept with much heuristic value is that psychophysiological changes reflect changes in arousal states or levels (i.e., Pinneo, 1961; Schnore, 1959). However, there is a major argument against the acceptance of a general dimension of activation or arousal when it is measured by peripheral physiological variables. The correlations between the different physiological measures have often been found to be low, both for base levels (resting state) and for the degree of change from low stress to high stress conditions for different individuals (Lacey & VanLehn, 1952).

On the other hand, it has been found that within individuals the changes in these variables are fairly reliable both when the same stressor is repeated (Lacey and VanLehn, 1952) and when reactions to different stressors are observed (Schnore, 1959). Nevertheless the idiosyncratic patterning of an individual's responsivity on different physiological measures indicates that there are problems in using these measures to assess differences in a general arousal state between individuals.

Rickles (1972) provides abundant electrophysiological and lesioning evidence for the existence of discrete but interactive neural systems which are differently related to the various psychophysiological response systems (such as heart rate, blood pressure, vasomotor changes and electrodermal responses). Furthermore, the facilitation or inhibition within these systems does not always result in a one to one relationship with measures of somatomotor activity. One certainly cannot take one measure, for example, heart rate, and assume that because one individual has a higher heart rate than another this difference is a good measure of a relatively general arousal state. The reverse order may be found on other variables. Levels on physiological measures are obviously not determined solely by a single factor that has a general effect. Among other determinants these levels are influenced by anatomical and physiological differences between individuals and type of orientations or "set" toward a situation, in addition to variations in factors that seem to influence more diffuse activation. An example of this is seen in the demonstration of decreases as well as increases in electrodermal activity of normal subjects to a cold pressor task (immersion of a limb in ice water) in spite of the fact that other psychophysiological measures such as heart rate and EMG increase in all subjects as a result of the high stressing effects of the cold pressor task. (For a review, see Lovallo, 1975). For the subjects in the above example it is necessary to employ some sort of additional concept, such as differences in "set" or orientation between individuals to account for the "paradoxical" electrodermal effects.

In a review of the literature using electrodermal activity as an index of arousal in schizophrenics Depue and Fowles (1973) reported

that previous studies have found no consistent differences between schizophrenics and normals although other psychophysiological measures have shown chronic schizophrenics to be more aroused. The authors report that a more detailed look at the electrodermal research, however, reveals that consistent results have been reported for two aspects of electrodermal activity: (1) habituation of responses or levels during periods of minimal stimulation, such as tones or lights of low intensity, was slower in schizophrenics and (2) frequency of spontaneous electrodermal responses was higher regardless of stimulus conditions. The authors concluded that both of these measures appeared to reflect arousal and that chronic schizophrenics are over-aroused (Depue & Fowles, 1973).

Jordan (1974) has reviewed a number of reports which challenge the generality of the conclusions of Depue and Fowles (1973). Jordan concludes that more generally, there is evidence of abnormally low electrodermal activity and responsiveness among some schizophrenics. He also argues that there is evidence of systematic trends in the behavior of such patients paralleling their physiological abnormalities. He concludes that theories of schizophrenia need to account for the occurrence of hyperelectrodermal, hypoelectrodermal, "paradoxical" electrodermal reactivity, and tonic activity in connection with the disorder.

Gruzelier and Venables (1972) in a study of habituation of electrodermal reactivity during periods of minimal stimulation (habituation to repeated tones with minimal background noise) in medicated schizophrenics found that the schizophrenics fell into two categories, regardless of length of hospitalization. On the basis of whether electrodermal

reactivity occurred to the repeated tones, one group of schizophrenics termed "responders" had high amplitude SCRs to the tones which did not habituate. The other group of schizophrenics, termed "nonresponders" showed no electrodermal reactivity during this tones habituation task. The electrodermal reactivity of the control subjects habituated over trials. The "responders" whether short term or chronic patients, support the evidence reviewed by Depue and Fowles (1973). The "nonresponders" whether short term or chronic, on the other hand, support the evidence reviewed by Jordan (1974). Medication and specific sub-diagnosis (i.e., paranoid, catatonic, chronic undifferentiated) did not differ between the two groups of schizophrenics. Number of spontaneous fluctuations for the short term responders was significantly higher than that of the control group; however, the number of spontaneous fluctuations in the chronic responders, while in the same direction, only approached significance, compared with controls. The nonresponders, on the other hand, both chronic and acute, had significantly fewer spontaneous fluctuations compared with normal controls. The electrodermal levels of both acute and chronic responder schizophrenics were higher than the levels of the controls; however, only the levels of the acute patients reached significance statistically. The electrodermal levels of both acute and chronic nonresponders were significantly lower than those of the control group. The generalization presented by Jordan (1974) that behavioral evidence parallels the psychophysiological evidence is also supported by the 1972 Gruzelier and Venables study. They reported that one of the few diagnostic differences between responders and nonresponders occurred with the classification of chronic schizophrenia with florid symptoms, and defect or residual schizophrenic states, there being

only responders in the former category and mostly nonresponders in the latter.

Impressed by the lateral asymmetry of electrodermal orienting responses reported by Luria and Homskaya (1963) in instances of unilateral brain damage and the tendency of bilateral differences in groups of schizophrenics reported by Dykman, Reese, Galbrecht, Ackerman, and Sunderman (1968), Gruzelier (1973) attempted to gather evidence for Flor-Henry's (1969) hypothesis by replicating the Gruzelier and Venables (1972) study with the addition of bilateral measures of skin conductance activity. Schizophrenic group differences reported in their 1972 study were replicated for electrodermal levels, spontaneous fluctuation frequencies, and SC orienting response characteristics, including latency and recovery time, amplitude, habituation characteristics of response amplitudes, and SC levels. In addition, bilateral asymmetry of SCR was reported for some of the response measures. A reduction or absence of responses was observed in the institutionalized (chronic) schizophrenic responders in the direction of less reactivity for the left hand. Marked bilateral differences in the levels occurred in both groups of schizophrenics but in opposite directions. The levels were higher in the responder group for the right hand and higher in the nonresponder group for the left hand.

Gruzelier and Venables (1973, 1974) using one of Luria and Homskaya's (1966, 1970) simpler instructional paradigms attempted to differentiate temporal and frontal dysfunction in what appears to be the same medicated schizophrenics used in the Gruzelier 1973 study. Luria and Homskaya (1966, 1970) had reported that the effect of instruction on the autonomic components of the OR were related to both the site and

the degree of brain damage. Verbal instruction increased the "regularization" of autonomic components in patients with temporal and parietal lesions but not in frontal patients whose damage was severe (see previous section). Gruzelier and Venables (1973, 1974) found that when SC responses were studied in an attentional discrimination task (subjects pushed a button to a previously habituated tone but not to a new tone) schizophrenic nonresponders behaved like brain damaged patients with lesions outside of the frontal areas. Most of the schizophrenic nonresponder patients in this task exhibited electrodermal reactivity to signal tones and remained nonresponders only to the neutral tones. A few nonresponding schizophrenics remained nonresponders to both tones. The schizophrenic responders, on the other hand, maintained electrodermal reactivity to both the signal and the neutral tones and the frequency of SCRs to the neutral tones was significantly higher than that found for those schizophrenic nonresponders who were exhibiting reactivity to the signal tones.

Differences between control groups and psychopathological groups have also been reported by a number of researchers for two other electrodermal measures not discussed previously. Mednick and Schuslinger (1968) reported shorter latencies and shorter SCR recovery measures of phasic SCRs in high risk (schizophrenic mothers) children who became sick compared with high risk children who did not become sick and healthy control (normal mothers) children. Shorter SCR latencies (Gruzelier, 1973; Gruzelier & Venables, 1972) and shorter one-half amplitude recoveries (Ax & Bamford, 1971; Gruzelier, 1973; Gruzelier & Venables, 1972) were reported in responding schizophrenics compared with controls during simple stimuli such as repeated tones presentations.

The shorter SCR latencies in responding schizophrenics relative to control subjects tended to disappear in more complex stimulus situations such as a tones discrimination task (Gruzelier & Venables, 1973). Broen (1968) reviews evidence for this dichotomy of responding during simple versus complex tasks. During simple behavioral tasks, the differences between schizophrenics and normal controls on latency of behavioral responding is slight and often nonsignificant; however, with increases in task complexity, the increase in latency of behavioral responding is much greater in the schizophrenics than in the controls, becoming significantly longer. The behavioral data suggests then, that the shorter SCR latencies of the acute schizophrenics, compared with controls, should increase more in this group with increasing task complexity than in the control group.

It is not known whether the shorter recovery measures of responding schizophrenics compared with controls in simple stimulus situations would disappear during complex tasks, since normal control subjects were not used during both simple (Gruzelier, 1973, 1974; Gruzelier & Venables, 1972) and complex tasks (Gruzelier & Venables, 1973). The work of Edelberg (1970, 1972b) suggests that the differences in recovery measures would disappear during more complex tasks. Edelberg (1972b) demonstrated that the recovery time of phasic SCRs is mediated by the CNS and is shortest during "goal-oriented" tasks such as mirror trace, arithmetic problems and reading outloud. The recovery times are longer during both spontaneous fluctuations and SCRs during defensive situations such as threat of shock.

Information on the latency and recovery times of nonresponding schizophrenics is, at best, sketchy or not available since these

patients did not respond (Gruzelier, 1973, 1974; Gruzelier & Venables, 1972, 1973) during simple stimulus situations. However, the data presented in these studies for nonresponding schizophrenics tends to suggest that their latencies and recovery times are longer than those of the responding schizophrenics.

Purpose of the Study

Several studies (Ax & Bamford, 1971; Bernstein, 1970; Gruzelier, 1973; Gruzelier & Venables, 1971, 1972, 1973, 1974; Mednick, 1970; Mednick & Schuslinger, 1968; Stern, Surphlis & Koff, 1965; and Venables, 1972) have demonstrated that psychopathological groups differ with respect to a number of psychophysiological response measures compared with controls.

In the present study an attempt was made to determine the nature of psychophysiological response differences in a variety of test situations of varying task complexity expected to alter the autonomic components of the orienting reaction. For the simpler stimulus situations the study is similar to the previous work of Gruzelier and Venables (Gruzelier, 1973, 1974; Gruzelier & Venables, 1972, 1973) except that a more restrictive criteria was used in testing for a bimodal distribution of electrodermal response characteristics of schizophrenics. Secondly, an assessment of bilateral differences for many of the electrodermal response measures was made to investigate the significance of laterality effects in these response measures; such effects have been reported previously but infrequently. Finally, the introduction of tasks of varying complexity provides a stronger test for generalizing from earlier findings which demonstrated differences between schizophrenics and normals on several electrodermal response measures but only in

simple stimulus situations.

METHOD

Subjects

The subject population consisted of two groups: selected schizophrenics and a normal control group.

Normal Control Subjects. The normal control group consisted of 17 dextral male volunteers. These included medical interns, staff psychiatrists, nurses, psychologists, students and ward orderlies. Care was taken to insure that a wide range of age and level of intellectual functioning was sampled. All of the volunteers were screened with the neuropsychological test battery to rule out the presence of significant cerebral dysfunctioning. Subjects with a history of head injury and/or loss of consciousness for extended periods were excluded. An attempt was made to collect intelligence quotients for all subjects.

Schizophrenic Subjects. The psychiatric diagnosis of the patients tested was made independently by two psychiatrists on the staff at AHE who allocated patients into the schizophrenic category only if, inter-alia, they presented with Schneiderian symptoms[1] of the first rank (Schneider, 1959). More generally the diagnostic criteria employed were those advocated by Feighner, Robins, Guze, Woodruff, Winokur, and Munoz[2] (1972). The mental state of the patient was documented by the Wing Mental State examination schedule (Wing, Birley, Cooper, Graham & Isaacs, 1967). No attempt was made to control for sex, handedness, length of hospitalization or medication.

Patients from the "long stay" wards and newly admitted patients were included in the sample if they met the following requirements: (1) concurrence of the diagnosis of schizophrenia by two psychiatrists

after one week of observation, and again, several weeks after the initial agreement as to diagnosis was reached, and (2) they were not considered to be mentally defective. An attempt was made to collect neuropsychological and I.Q. functioning data on all the patients included in the schizophrenic group.

A total of 24 patients were tested. Of these, seven were excluded from the study when a lack of agreement as to the diagnosis occurred after several weeks of observation, and two were excluded due to uncooperativeness during the testing sessions.

The remaining 15 schizophrenic subjects were divided into two groups of nearly equal size on the basis of recordings of their electrodermal activity during performance of a task, much like the method described previously by Gruzelier and Venables (1972). The difference between the criteria for forming subgroups of schizophrenics employed by Gruzelier and Venables (1972) and the method used in the present study was the task used to differentiate responder from nonresponder schizophrenics. Gruzelier and Venables (1972) labeled all schizophrenics with electrodermal reactivity to repeated presentations of tones during the habituation task as responders. Schizophrenics with no electrodermal reactivity during the presentation of these tones were called nonresponders. In the present study schizophrenics with electrodermal reactivity during cognitive tasks such as arithmetic problems or spelling backwards problems were designated responders. Schizophrenics with no electrodermal reactivity or minimal reactivity (less than .04 μmho criteria) during the performance of such cognitive problems were labeled nonresponders.

The necessity for abandoning the criteria employed by Gruzelier

and Venables (1972) became evident in pilot work at AHE. Examination of control subjects at AHE resulted in three populations when using the criteria employed by Gruzelier and Venables (1972); the three groups consisted of normals who did not habituate electrodermal reactivity to repeated tones (i.e., responders), normals who responded initially but habituated electrodermal reactivity to repeated tones (i.e., habituators), and normals who showed no SCRs to repeated tones (i.e., nonresponders). Furthermore, the electrodermal reactivity of schizophrenics was not congruent with that of Gruzelier and Venables' schizophrenics during the presentation of repeated tones. That is, some schizophrenics responded but habituated their initial reactivity to tones presentation. More importantly schizophrenics were observed with "paradoxical" reactivity as described by Jordan (1974). These schizophrenics showed no reactivity during attentional or cognitive tasks and yet showed reactivity during some of the repeated tones trials. Gruzelier and Venables (1974) themselves report similar data in that a few subjects in the groups of depressives and personality disorder groups fell into the responder (no habituation) and nonresponder categories, although the majority of their subjects in these two groups exhibited responses which habituated quickly (Gruzelier & Venables, 1974). Similarly, they reported that seven out of 100 normals tested fit into the nonresponder category (Gruzelier & Venables, 1972) although they made no mention of any normals who fit into the responder category (on the basis of a lack of habituation to repeated tones). Since the normals observed at AHE in several studies always demonstrated some electrodermal reactivity during cognitive tasks, schizophrenics were differentiated into responder and nonresponder categories in the present study on the basis of their

electrodermal reactivity during the performance of cognitive tasks only.

Age and sex of the schizophrenic and control subjects and length of hospitalization of the schizophrenic subjects are summarized in Table 1.

The type and daily amount of drug intake by subjects in the schizophrenic group is summarized in Table 2. This table includes an equivalency "scale of sedative effects" of the medication for all major tranquilizers based upon the following ratios: 50 mg Chlorpromzaine = 1 unit, Nozinan 1:1; Stelazine 1:10; Trilafon 1:12.5; Mellaril 1:1; Moditen 1:168; and Haldol 1:10.

Equipment

All psychophysiological recordings were taken in a quiet room maintained at a temperature between 22°C ± 2°C during the spring and summer of 1975.

A Beckman type R Dynograph was used to record heart beats, respiration rate, and bilateral electrodermal activity. Heart beats were recorded via electrodes taped to the chest (by modified lead II placements) and the signal fed into a Beckman type 9854 heart rate monitor coupler. Respiration rate was recorded with two mercury strain gauges, one placed around the upper chest and the other placed around the chest at the level of the diaphragm. The signals from the two gauges were connected in parallel and fed into a Beckman type 9853 A coupler. Bilateral skin conductance was measured from bipolar placements from the distal phalanges of the first and second fingers of both hands. Beckman biopotential electrodes (silver chloride pellets) and Beckman masks (1.016 cm diam) covered a skin area of 0.8107 cm^2 under each electrode. All SCR electrodes were aged in a .5% potassium

TABLE 1

Mean ages of the two schizophrenic groups and control group, mean current length of hospitalization, number of previous known hospitalizations and mean length of total hospitalization for both schizophrenic groups.

		Schizophrenic Responders	Schizophrenic Nonresponders	Controls
N		7 males	6 males, 2 females	17 males
Age (years)	$\bar{X}$	30.1	35.6	30.3
	σ	3.9	9.5	9.9
	range	24-36	20-51	19-53
Length of Current Hospitalization (wks)	$\bar{X}$	5.6	25.2	
	σ	5.3	3.7	
	range	1-17.3	5-69	
No. of previous Hospitalizations	$\bar{X}$	3.7	6	
	σ	2.3	2.4	
	range	1-7	3-9	
Total Duration of Hospitalization (yrs)	$\bar{X}$	2.4	6.7	
	σ	3.3	4.6	
	range	.02-9.8	1.5-14.9	

TABLE 2

Type of and daily drug doses in mg. An equivalent table for the sedative effects of major tranquilizers only follows the drug dosage. Each equivalency unit equals 50 mg chloropromazine. An ST after amount of drug refers to meds started within 1-5 days.

Responders				Nonresponders			
S#	Medication	Daily Dose	Equiv. Units	S#	Medication	Daily Dose	Equiv. Units
1	Moditen	3.57 mg.	12.0	1	Trilafon	60 mg. (ST)	15.0
2	Largactil	1200 mg.	24.0	2	Trilafon	24 mg.	6.0
3	Unmedicated		0.0		Valium	20 mg.	
4	Nozinan	75 mg.	7.5	3	Haldol	40 mg.	8.0
	Haldol	30 mg.		4	Nozinan	100 mg.	14.0
5	Stelazine	60 mg.	14.0		Moditen	3.57 mg.	
	Largactil	100 mg. (ST)		5	Mellaril	100 (ST)	2.0
6	Stelazine	40 mg.	8.0	6	Trilafon	48 (ST)	12.0
7	Trilafon	48 mg. (ST)	12.0	7	Unmedicated		0.0
	Elavil	50 mg. (ST)		8	Largactil	1200 mg.	40.0
					Stelazine	80 mg.	
					Elavil	225 mg.	

chloride solution. Just prior to the "hookup" of each subject the battery potential of each pair of SCR electrodes was determined and only those pairs showing a potential of .1 millivolt or less were used. The biopotential electrodes were filled with a .5% potassium chloride paste (2 gm agar in 100 ml of .5% potassium chloride solution continually stirred until cool: see Lykkens & Venables, 1971). Skin conductances were recorded directly with constant voltage systems (Beckman 9844 skin conductance couplers) feeding two channels of the Beckman Dynograph. Sensitivity ranged between .5 μmho/cm and .2 μmho/cm.

Experimental Procedures

Prior to testing all of the subjects received an explanation of the basic test procedure and questions concerning the procedure were answered. In the case of hospitalized patients permit forms were signed before the procedure began.

Just prior to testing all subjects washed their hands thoroughly with an alkaline soap and then were seated in a reclining chair for the duration of the testing session. The recording equipment was located in the same room as the subject but was screened from view.

After the electrodes were placed subjects were allowed to sit quietly for ten minutes to allow for hydration of the SCR electrolytic paste. Then ten minutes of recordings were made to collect personal history data from the subject. During the next hour of this session, eight experimental tasks were carried out before the subject was unhooked from the apparatus. The eight tasks employed are described below.

Task 1. Physical Stimulation of the Autonomic Nervous System. The subject was instructed to inhale fully and then exhale as rapidly as possible upon command from the experimenter. This response was

repeated three or four times at 30 to 60 sec intervals. The subject was then asked once to inhale and hold his breath until asked to release it. Finally the subject was asked to cough once as hard as possible upon command from the experimenter. Coughing was induced several times upon instruction by the experimenter at 30 to 60 sec intervals.

Task 2. Tone Habituation and Dishabituation Task. The subject was fitted with Sennheiser open air HD 414 headphones and asked to listen to the following instructions through the headphones: "You are going to hear a series of tones. Please sit quietly with your eyes open and listen to them." The subject was then presented with 18 tones of two second duration at 93 dB ± 3 dB occurring at pseudo-random intervals of between 20 and 75 seconds ($\bar{X}$ interval = 45 seconds). The higher (93 dB) intensity in the open air headphones was subjectively equivalent to an intensity of 80 dB from a sealed air type of headphone (Braun KH 1000). All intensity measurements were made using a Brüel and Kjaer 4153 artificial ear. The Sennheiser foam cushions were removed during intensity measurements.

The first 15 tones (habituation phase) were 1000 Hz signals followed by three tones at 2000 Hz (dishabituation phase). The subject was then asked how many tones he had heard and whether or not he had counted them in order to exclude data from subjects who had introduced self-imposed attentional value to the tones. Next Task 1 was partially repeated: the subject was asked to take 1-5 deep breaths at 30 to 60 second intervals.

Task 3. Tone Attentional Task. A foot switch mounted on a pedestal fastened to the floor was adjusted such that the subject's right foot was fully extended when the subject had the heel of his

right foot resting on the heel plate of the switch. The subject was then instructed to operate the button on the foot switch by gently depressing the plate under the ball of his right foot. He was informed that he would be required to perform this response (depressing button) during the next task. The following instructions were then presented through the headphones:

> Next, you will hear two different tones at different times. When you hear this tone (sample 1000 Hz presented) please press the button. However, when you hear this tone (sample 2000 Hz presented) do not press the button. Remember, press the button each time you hear the following tone (1000 Hz sample presented) and do not press the button when you hear this tone (2000 Hz sample presented).

Tones of 93 db, $\pm$ 3 of two second duration pseudorandomized and occurring at an interval of 15 to 90 seconds ($\bar{X}$ interval = 30 seconds) were then presented through the headphones. If the subject was unable to perform the task correctly he was given verbal feedback by the experimenter indicating which responses were incorrect until a criterion of six correct responses had been made. Once the criterion was reached the presentation of the tones was continued until a criterion of 12 correct responses to each tone had been made. Occasionally patients had to be coached more than once in order to establish the correct response consistently and before they could complete the task requirements. Some subjects refused to perform the task; in these instances the task was terminated, the headphones were removed and the subjects were told

to "relax for a few minutes".

Tasks 4 and 5. Cognitive Demands. The skin area of the subject's right calf muscle was rubbed with an abrasive paste (EKG) and a Tursky (1973) concentric electrode was attached with a strip of Velcro and an elastic band. The electrode was connected to a well isolated shock source (Farrell instruments) with an intensity range varying from zero to 1 ma. Subjects were told that the electrode was a "stimulus" which would be used "in about five minutes". Subjects were allowed to continue sitting for a few minutes following electrode placement on the leg until an acceptable level of SC activity was reached (in the same general range as was observed for Task 3 as a "resting level"). Then the subject received the following instructions for Task 4: "Next, I will give you a series of arithmetic problems. Please do them as quickly as you can. Once you have given me an answer I will give you the next problem until we have finished the series." The experimenter began the arithmetic series 30 seconds after completing the instructions. The arithmetic series consisted of the following problems: 12 + 4; 27 x 4; 7 + 16; 3 x 17; 89 + 12, and occasionally, 119 + 7. If the first one or two problems seemed to be too difficult for the particular subject to perform, a more simple series was employed consisting of simple addition such as 9 + 3.

Approximately 30 to 90 seconds after finishing the arithmetic problems, the subject was given instructions for Task 5: "Next, I will spell words backwards to you. You are to turn the letters around as quickly as you can. Once you have given me an answer, I will give you the next problem until we have finished the series." After a 30 second pause the experimenter presented the first word spelled backwards. The

following words were used: bush, cone, filth, judge, and navel.

Task 6. Mirror Tracing Task. Subjects were handed the LaFayette Tracing Kit and instructed on how to perform the task. The subjects were told to "try in a different place" if they got "stuck". Subjects were told to begin the task 30 to 60 seconds after the instructions were given. The task was terminated if not completed within ten minutes.

Task 7. Stressor Response. Subjects were re-fitted with the headphones used in the earlier tasks. They were informed that two different tones would be presented at five second intervals for each trial. During the presentation of the second tone they were told to tell the experimenter when they first felt a sensation in the leg which had the circular electrode. The current from the shock source was gradually turned up. Most of the subjects reported a tickling sensation at .1 ma, although some did not report any sensation until they reached .15 ma. The subject was then told that the experimenter would turn up the intensity until the subject told him to stop. Once this maximum intensity was determined by the subject, several SCRs were recorded during the presentation of shock at this maximum intensity. Then the shock electrode and the headphones were removed.

Task 8. Startle Response. Subjects were told that the session was finished, asked to shut their eyes and relax as much as possible, to inhibit all movement and to keep their eyes shut until the experimenter told them to open their eyes. After the subject had complied with these instructions for two or three minutes a sudden loud noise, produced by a large object being dropped on the floor close to the subject's head occurred and the behavioral reactions as well as the psychophysiological

responses were recorded.

Response Definition and Data Quantification

The total possible number of SCR and HR responses scored for each subject was 68 and 58, respectively. Forty-nine SCR and HR responses were simultaneous events, time locked to the onset of a stimulus. The remaining 19 SCR and 9 HR responses were selected for analysis on the basis of the criteria listed below.

Heart Rate. Each of the scored HR responses were blocked into 15 second pre-stimulus periods and 30 second post-stimulus periods, with the post-stimulus period time locked to the stimulus onset. For the nine selected HR responses, four consisted of the post-stimulus period occurring at the onset of instructions and performance of problems for the arithmetic and spelling backwards problems and two HR responses during an arbitrary midpoint of the 30 second waiting periods between the presentation of instructions and problems. The three remaining HR responses which were recorded occurred during the start of mirror tracing problems, an arbitrary midpoint, and the period immediately following termination of the mirror trace task.

The inter beat intervals (IBI's) within each blocked 15 and 30 second period were read on an IBM OSCAR SCANNER, stored on 9 track tape, played back for verification of digitizing accuracy, labelled and stored on 9 track tape.

Definition of an HR response consisted of a pre-stimulus baseline determination on the basis of the first 10 beats prior to stimulus onset (BL:HR). Next, the shortest IBI, representing the highest HR, was determined within the first 6 beats after the stimulus onset (H:HR). The longest IBI, representing the lowest HR was determined within the

first 20 beats after stimulus onset (L:HR). The L:HR minus H:HR change scores (L-H:HR) were then calculated (see Lang & Hnatiow, 1962). In order to determine the relative contribution of the H:HR and L:HR scores to the change scores, the baseline was subtracted from the slowest HR (L-B:HR) and the fastest HR response was subtracted from the baseline (B-H:HR). The sequential positions with respect to the post-stimulus onset of each H:HR and L:HR were also scored in order to determine the shape of the HR response.

Electrodermal Activity. The bilateral electrodermal activity was scored manually using a template etched on thin (1/8th inch) plexiglas to minimize parallax. A variety of response measures were used to assess electrodermal activity. Amplitude of SCRs was defined as the height of the SCR peak above SC level and converted into μmho values. All SCRs .01 μmho or larger were scored; however, those SCRs .04 μmho or larger were coded in order to assess the data with amplitude criteria similar to those employed by Gruzelier and Venables (1972).

On all trials requiring no motor response from the subject phasic SCRs were defined as the first pen excursion occurring .5 to 5 seconds after the stimulus onset. On trials requiring a motor response the phasic SCR was defined as the first pen excursion occurring 0 to 5 seconds after the motor response.

The latency of all phasic SCRs was scored as the time elapsing between the stimulus onset and the onset of the SCR. Ascent time was defined as the time elapsing from SCR onset to peak of the SCR curve. Recovery time was defined as the time elapsed from the peak of SCR curve to a value of 50% less than the peak amplitude value. For each scored SCR a baseline SC level was determined from the latency period

prior to each SCR.

The selection procedure for the 18 scored SCRs which were not time locked to a stimulus event consisted of the following criteria. Up to two separate spontaneous fluctuations were selected and scored for all electrodermal measures except latency. These spontaneous fluctuations were simply arbitrary choices of the experimenter taken from the population of spontaneous fluctuations (over .04 μmho amplitude) counted during the habituation/dishabituation period. Care was taken to exclude from the frequency count of spontaneous fluctuations any SCRs occurring up to 10 seconds after tone onset as well as any SCRs occurring during body movements or respiration rhythm changes (i.e., yawning).

During arithmetic and spelling backwards instruction periods, 30 second waiting periods and problem solving periods the largest amplitude SCRs were frequently followed by other SCRs; recovery time was seldom available. Therefore, a second SCR was selected within each period providing it met the following criteria: (a) recovery time was available, (b) SCR was not the result of a verbal report by the subject, and (c) SCR was not the result of body movements or large respiration rhythm changes. The SCRs during mirror trace task instructions and problem solving were also selected according to these criteria. All manually scored responses were then key-punched and verified on IBM cards.

Psychological Assessment of Subjects

All subjects in the present study were referred to the Neuropsychological Department at AHE for psychological assessment at approximately the same time that the subjects were tested psychophysiologically. The WAIS was administered to assess the intellectual functioning and a modified version of the Halstead-Reitan Neuropsychological Battery was administered to assess the presence of brain dysfunctioning.

For a normal subject, the combined procedures lasted about 4 and 1/2 hours; for a very dysfunctional subject the procedure required as long as four days for testing.

Filskov and Goldstein (1974) compared the diagnostic validity of the Halstead-Reitan Neuropsychological Battery with the more popular physical diagnostic techniques (brain scan, angiograms, EEGs, and so on). Using a clinical-actuarial approach in interpreting the Neuropsychological Battery, they reported extremely high "hit rates" in assessing the presence of brain dysfunction (100%) and the lateralization and neuropathological process involved (89%). Filskov and Goldstein's Halstead-Reitan Battery contained seven separate variables whereas the modified version of this same battery used at Alberta Hospital, Edmonton contains 32 variables (see list of variables in Appendix F).

RESULTS

Experimental Group Differences

In the present study with no control for length of hospitalization, responders and nonresponders were found with approximately equal frequency (seven responders, eight nonresponders). A Mann-Whitney U comparison of the length of current hospitalization (see Table 1, method section) between responders and nonresponders revealed no significant differences, U (7,8) = 27.5, p = .50. However, the responder-nonresponder selection criteria utilized in the present study did correlate with length of illness: nonresponders were found more frequently among those patients with a longer history of illness. The number of previous hospitalizations was less for the responders and this difference approached significance, U (7,8) = 14, p = .06. The total length of previous hospitalizations was less for the responders than for the nonresponders, U (7,8) = 8, p = .01.

In the present study the dosage levels of the major tranquilizers did not differ between the responder and nonresponder groups. Unfortunately it was not possible to equate the sedative effects of other drugs which some of the patients were receiving. The daily dosage level for each patient of a major tranquilizer was converted into an equivalency unit according to the ratios listed in the methods section (see Table 2). A Mann-Whitney U comparison between groups of the dosage equivalency units did not yield any significant differences, U (7,8) = 27.5, p = .50.

A comparison of the average daily doses of the major tranquilizers received by the patients in the Gruzelier and Venables (1972) study was made by converting these daily doses into the same equivalency units described above. This conversion resulted in much smaller units than

those in the present study: 7.9 for responders and 6.0 for nonresponders compared with 12.9 for the responders and 13.9 for the nonresponders, respectively in the present study. Thus the average of the medication doses given to the patients in the present study was much heavier than that given to the patients in the Gruzelier and Venables study (1972).

In order to further assess drug effects on differences between responders and nonresponders, rank order correlations were performed between electrodermal levels, frequency of spontaneous fluctuations, and levels of medication since SC levels and spontaneous fluctuations are most frequently reported as being reduced by the major tranquilizers (Tecce & Cole, 1972). Minimal correlations (Table 3) were observed in the combined schizophrenic population in the present study (see also Appendix G)

Skin Conductance Response Frequencies

Skin conductance response frequencies were scored during the habituation/dishabituation task, the tones attentional task, as well as a count of the spontaneous fluctuations during the habituation/dishabituation task. The amplitude value used to count SCRs (.04 μmho) was chosen to provide the same sensitivity utilized by Gruzelier and Venables (1972) after taking into consideration the surface area of the skin under consideration in both the present study and that of Gruzelier and Venables (1972).

Habituation/Dishabituation (Task 2). Figure 1 illustrates the percentage of subjects responding to each tone presentation. Nonresponders had significantly fewer responses during habituation and during habituation/dishabituation combined when compared to both the controls and responders (Table 4). The responders, however, did not differ significantly from the controls (Table 4). These results are in contrast to the results

TABLE 3

Spearman rank order correlations of drug dosages with electrodermal measures in the pooled (N = 15) schizophrenic population: Zero medication assigned the highest rank.

	r of Medication with SCL	r of Medication with frequency spontaneous fluctuations
Right hand	.33	.07
Left hand	.21	-.13

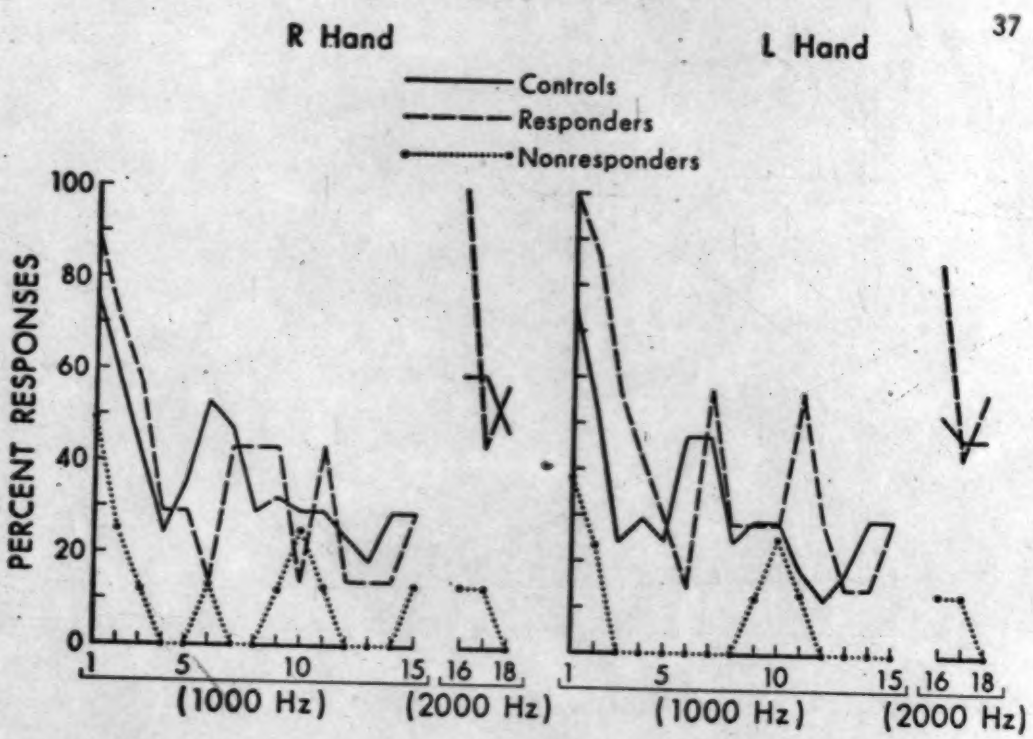


Figure 1. Percentages of subjects responding (.04 μmho criteria) for the three groups during habituation (1000 Hz) and dishabituation (2000 Hz) of task two.

TABLE 4

Mann-Whitney U values of both hands for the percentages of possible responses during the first 15 trials (habituation) of task 2 and during the entire 18 trials (habituation/dishabituation) of task 2 for all three groups.

	Responders (N = 7)		Nonresponders (N = 8)	
	Trials			
	1-15	1-18	1-15	1-18
Normals (N = 17)				
Right	56.5	55.5	30.5*	23.5**
Left	50.0	47.5	20.5**	20.5**
Responders				
Right			12.5*	5.5**
Left			7.0**	6.0**

One-tailed tests $p < .05$*

$P < .01$**

reported by Gruzelier and Venables (1972) who reported a significantly higher frequency of responding in schizophrenic responders, compared with controls, with no overlap in distribution of responding between these two groups. Pearson product moment correlations between amplitude of largest SCRs during cognitive tasks 4 and 5 and frequency of response during task 2 for the control group were nonsignificant. Right hand $\underline{r}$ (17) = .001, $\underline{p} > .05$; left hand $\underline{r}$ (17) = -.007, $\underline{p} > .05$. For the combined schizophrenic groups the correlations were, right hand $\underline{r}$ (15) = .24, $\underline{p} > .05$; left hand $\underline{r}$ (15) = .55, $\underline{p} < .03$. The schizophrenic scores on each variable were also converted to within group $\underline{z}$ scores and the $\underline{z}$ scores from both schizophrenic groups were then pooled. For the combined schizophrenic groups the correlations on the transformed scores were, right hand $\underline{r}_z(15)$ = -.18, $\underline{p} > .05$; left hand $\underline{r}_z(15)$ = .29, $\underline{p} > .05$.

Tones Attentional (Task 3). Figure 2 illustrates the percentage of trials with SCRs occurring during the signal and neutral tones of task 3. Nonresponders had significantly fewer responses during both the signal and neutral tones, compared with controls and responders (Table 5). In contrast, Gruzelier and Venables (1972) reported a significant increase in frequency of responding to the signal tones, with no responses during neutral tones in their nonresponding group. In the present study, no differences were found between the controls and responders, with respect to frequency of responding during task 3 (Table 5).

Pearson product moment correlations between SCR amplitudes during cognitive tasks 4 and 5, and frequency of responses during task 3 for the control group were nonsignificant for both the neutral (right hand $\underline{r}$ (17) = -.05, $\underline{p} > .05$; left hand $\underline{r}$ (17) = -.07, $\underline{p} > .05$) and

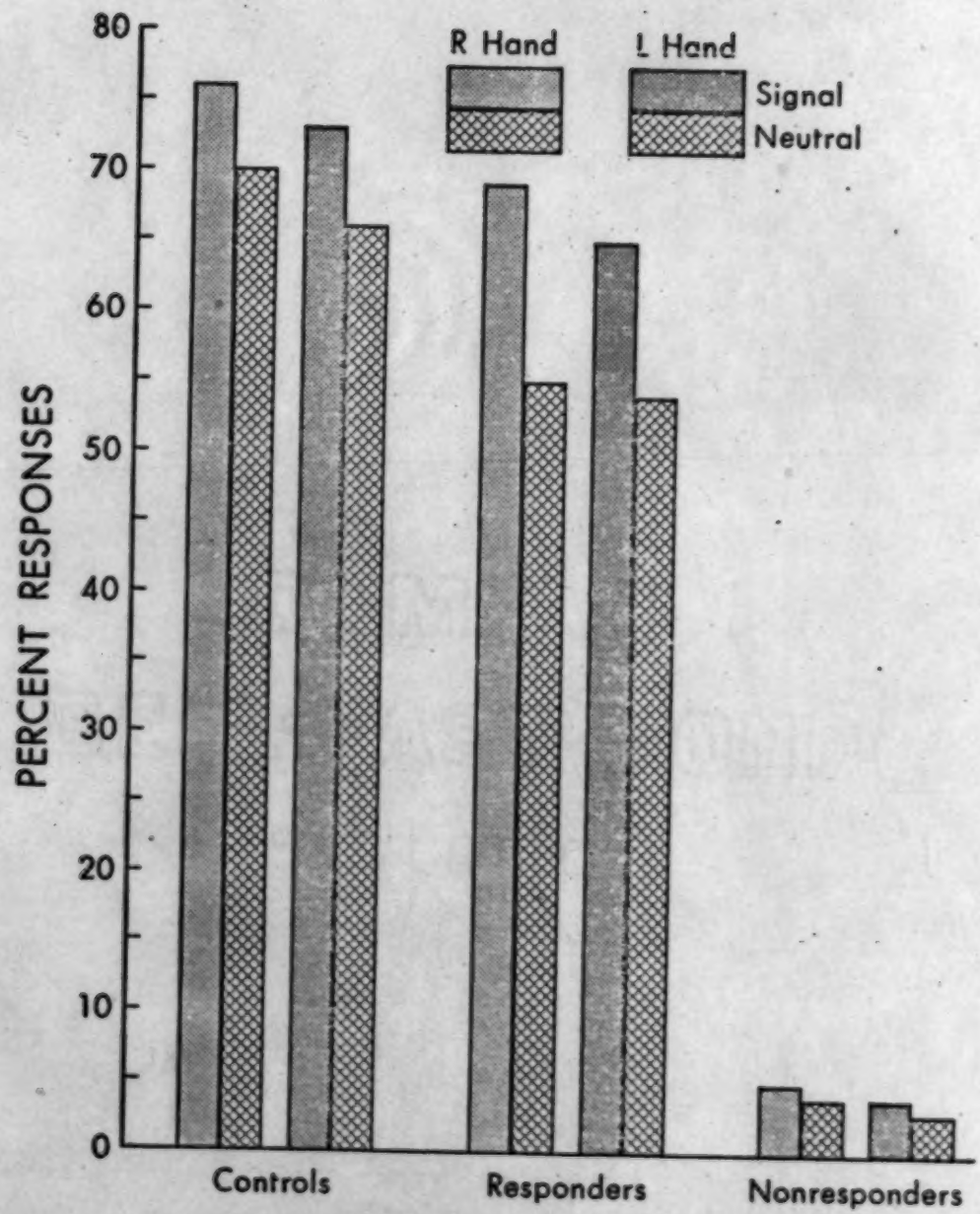


Figure 2. Percentage of trials with SCRs (.04 μmho criteria) occurring during the signal and neutral tones of task three for three groups.

TABLE 5

Mann-Whitney U values of both hands for the percentages of possible responses during the signal and neutral tones of task 3 for all three groups.

	Responders (N = 7)		Nonresponders (N = 8)	
	Signal	Neutral	Signal	Neutral
Normals				
Right	49	46.0	10.0***	4.0***
Left	50	51.5	6.0***	4.0***
Responders				
Right			2.0***	7.0**
Left			1.5***	4.0**

One-tailed tests $p < .05$*

$p < .01$**

$p < .001$***

signal (r (17) = -.35, p > .05; left hand r (17) = -.11, p > .05) tones. For the combined schizophrenic groups, the correlations were: neutral tones, right hand r (15) = .33, p > .05, left hand r (15) = .64, p < .01, and signal tones, right hand r (15) = .35, p > .05, left hand r (15) = .60, p < .02. For the combined schizophrenic groups the correlations on these variables, after being transformed to within groups z scores, were: neutral tones, right hand r (15) = -.01, p > .05, left hand r (15) = .46, p > .05, and signal tones, right hand r_z(15) = -.15, p > .05, left hand r_z(15) = .42, p > .05.

For both task 2 and task 3, no relationship appears between the amplitude of SCRs during cognitive tasks and the frequency of responses in the normal group. For the combined schizophrenic groups, a significant relationship appears, but only for the left hand. This relationship for the left hand does not disappear in the transformed correlations either, which were in the same direction and approaching significance.

Spontaneous Fluctuations. The mean spontaneous fluctuation frequencies are presented in Table 6 for the three experimental groups. Bilateral asymmetry of response frequencies were compared with correlated t tests but the comparisons failed to approach significance.

The schizophrenic responders had a higher incidence of responses than the control and nonresponder groups. Although the difference between the responders and controls only approached significance (right hand, Mann-Whitney U (7,17) = 3.5, p > .05; left hand, U (7,17) = 38, p > .05), the differences between the responder and nonresponder schizophrenics were significant, right hand U (7,8) = 6, p < .01; left hand U (7,8) = 5, p < .01. The nonresponders also had significantly fewer spontaneous fluctuations than did the control group, right hand

TABLE 6

Mean number of spontaneous fluctuations for both hands during the habituation/dishabituation task for all three groups.

		Right Hand		Left Hand	
Groups	N	$\bar{X}$	σ	$\bar{X}$	σ
Responder	7	29.1	28.8	37.6	38.0
Control	17	15.0	18.7	14.4	20.4
Nonresponder	8	4.0	5.4	2.6	4.1

U (8,17) = 34, $p < .05$, left hand U (8,17) = .34, $p < .05$. These results are consistent with those reported by Gruzelier (1973).

Pearson product moment correlations between the amplitude of SCRs during cognitive tasks 4 and 5, and frequency of spontaneous fluctuations were nonsignificant for both the control (right hand r (17) = -.16, $p > .05$; left hand r (17) = -.19, $p > .05$) and combined schizophrenic groups (right hand r (15) = .20, $p > .05$; left hand r (15) = .39, $p > .05$). The correlations between the criterion variable and frequency of spontaneous fluctuations remained nonsignificant after transformation to z scores, right hand $r_z(15) = -.34$, $p > .05$, left hand $r_z(15) = -.14$, $p > .05$.

Response Amplitude

Figure 3 summarizes mean amplitude differences between the three groups for all tasks except the habituation/dishabituation task. In computing mean amplitudes of SCRs zero amplitude values were included in the data. As Figure 3 illustrates, little difference in amplitude appears between the responders and controls, whereas the nonresponders differ for all the situations of Figure 3.

Tests of groups by situations effects of SCR amplitude were carried out on data for both hands with two-factor analyses of variance, with eleven repeated measures (the 11 situations of Figure 3) as one factor. The differences in mean amplitude between groups were significant for both hands, right hand F (28,29) = 16.48, $p < .001$; left hand F (28,29) = 11.56, $p < .001$. The repeated measures effect was also significant (right hand F (10,290) = 24.1, $p < .001$; left hand F (10,290) = 23.07, $p < .001$), as well as the group by repeated measure interaction (right hand F (20,290) = 4.31, $p < .001$; left hand F (20,290) = 4.73, $p < .001$).

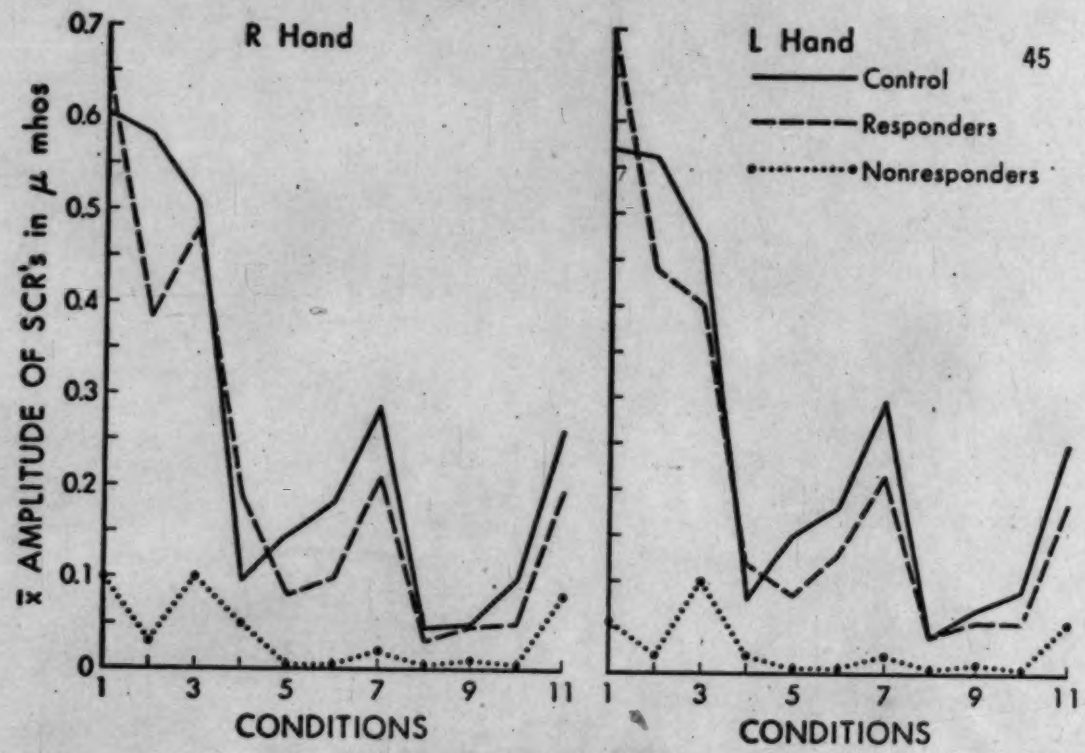


Figure 3. Mean amplitudes of SCRs for the R and L hand of the three groups during the following: (1) task one, physical stimulation, (2) partial repetition of task one after task two completed, (3) task eight, startle, (4) spontaneous fluctuations during task two, (5) neutral tones and (6) signal tones of task three, (7) largest amplitude SCRs during instructions and performance of cognitive tasks four, arithmetic, and five, spelling backwards, (8) SCRs, with recovery time available, during the instructions only of cognitive tasks four and five, (9) all SCRs, with recovery time available, during cognitive tasks four, five, and six, mirror tracing, (10) all SCRs, with recovery time available, during performance only of tasks four and five, and (11) task seven, shock.

Scheffe multiple comparisons ($p < .05$) for each situation of Figure 3 resulted in no significant differences of mean amplitudes between the normal control group and the responding schizophrenics (Table A, Appendix A). The nonresponders, on the other hand, had significantly smaller mean amplitudes for both right and left hands compared with controls for ten of the eleven repeated measures for the right hand and all of the repeated measures for the left hand (Table A, Appendix A). The nonresponders also differed significantly from the responders on several conditions of Figure 3 (Table A, Appendix A). Separate correlated t tests for each group showed that although all three groups demonstrated differences in mean amplitude between situations, this was much less frequent an occurrence for the nonresponder group (Tables B, C, D, Appendix A). Therefore, the significant groups by repeated measures interaction was interpreted as resulting from the larger mean SCR amplitudes and greater variation in SCR amplitudes across measures in both the control and the responding schizophrenic groups compared with that occurring for the nonresponding schizophrenics.

A detailed analysis of the SCR amplitude effects for all three groups during the habituation/dishabituation task (number 2) is presented in Appendix A. The difference in mean amplitude between groups only approached significance for one hand and was significant at the .05 level for the other hand.

The similarity of SCR amplitudes in the nonresponders compared to controls during stressless tasks such as habituation/dishabituation and the paradoxical attenuation of nonresponder SCR amplitudes during stressful tasks (i.e., task 8, startle) is illustrated in Figure 4. Only non zero amplitude responses are included in the mean SCR amplitudes

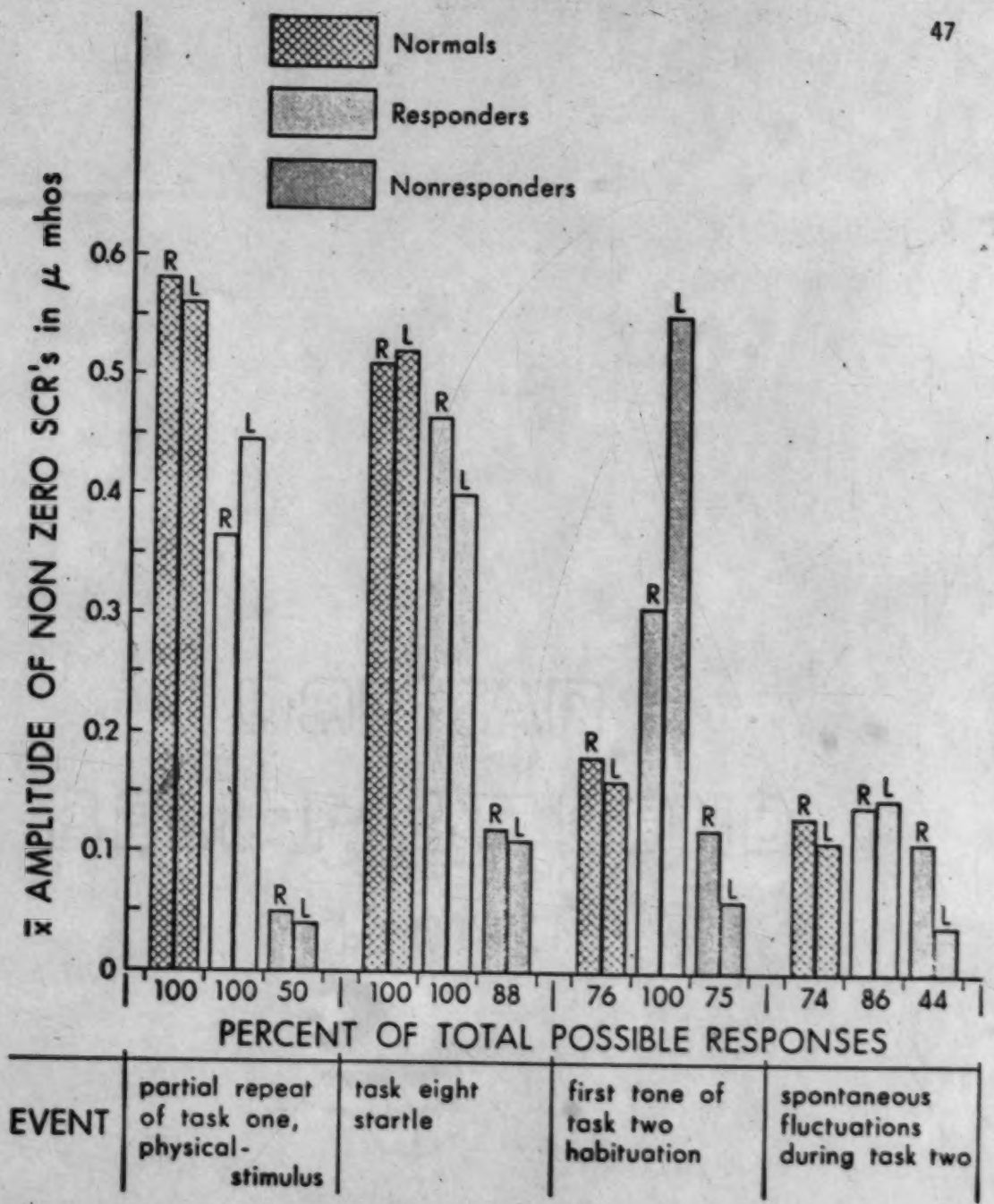


Figure 4. Mean amplitude of SCRs, with zero amplitude SCRs excluded, for three groups during four events.

of Figure 4.

Pearson product moment correlations between the amplitude of SCRs during cognitive tasks 4 and 5 and the amplitude of SCRs in other tasks is presented in Table 7. Pearson product moment correlations between the criterion amplitude variable (SCRs, tasks 4 and 5) and the SCR amplitude on other tasks, after these variables were transformed to within schizophrenic group *z* scores, are also presented in Table 7. Significant correlations for many of the tasks suggest a positive relationship between amplitude on the criterion task (for sorting schizophrenics) and amplitude on other tasks for both the control and schizophrenic groups. The evidence for a greater relationship of amplitude between tasks for the left hand, compared with the right, for both the controls and schiozphrenics is not understood.

The bilateral asymmetry of SCR amplitudes was also investigated in an attempt to confirm the significant SCR amplitude asymmetries reported by Gruzelier and Venables (1973). However, since no consistent SCR amplitude asymmetries were observed in more than two subjects in the entire study, further investigations were discontinued.

Response Latency

The mean response latencies for the right and left hand of all three groups are plotted in Figure 5 for all tasks with an SCR time locked to a stimulus onset. Latency differences between groups could not be analyzed with analyses of variance due to the frequent number of missing cells when an SCR did not occur. To determine if nonresponders were significantly slower in responding, independent *t* tests were computed for each task illustrated in Figure 5 between nonresponders and controls, and nonresponders and responders. The nonresponders were

TABLE 7

Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean SCR amplitude during other tasks. r_z = product moment correlations on these same variables after the within schizophrenic group raw scores were converted to z scores and then pooled for the correlations.

Task Number & Description	Controls				Schizophrenics					
	Right hand		Left Hand		Right Hand			Left Hand		
	N	r	N	r	N	r	r_z	N	r	r_z
1 Physical Stimulus	17	.32	17	.62**	15	.54*	.33	15	.34	-.01
2 Habituation/Dishabituation	17	.24	17	.23	15	.26	-.24	15	.56*	.33
3 Neutral Tone	17	.35	17	.58**	15	.44	.08	15	.62**	.54*
3 Signal Tone	17	.06	17	.43	15	.24	.06	15	.55*	.57*
4, 5, 6, SCRs with available recovery times	17	.58**	17	.69**	15	.84***	.59*	15	.82***	.46
7 Shock	15	.61*	15	.60*	14	.64**	.57*	14	.86***	.61*
8 Startle	17	.62**	17	.57*	15	.91***	.62**	15	.86***	.77***
2 Spontaneous Fluctuations	15	.11	15	-.03	13	.11	-.36	12	.26	-.38

$p < .05$*

$p < .01$**

$p < .001$***

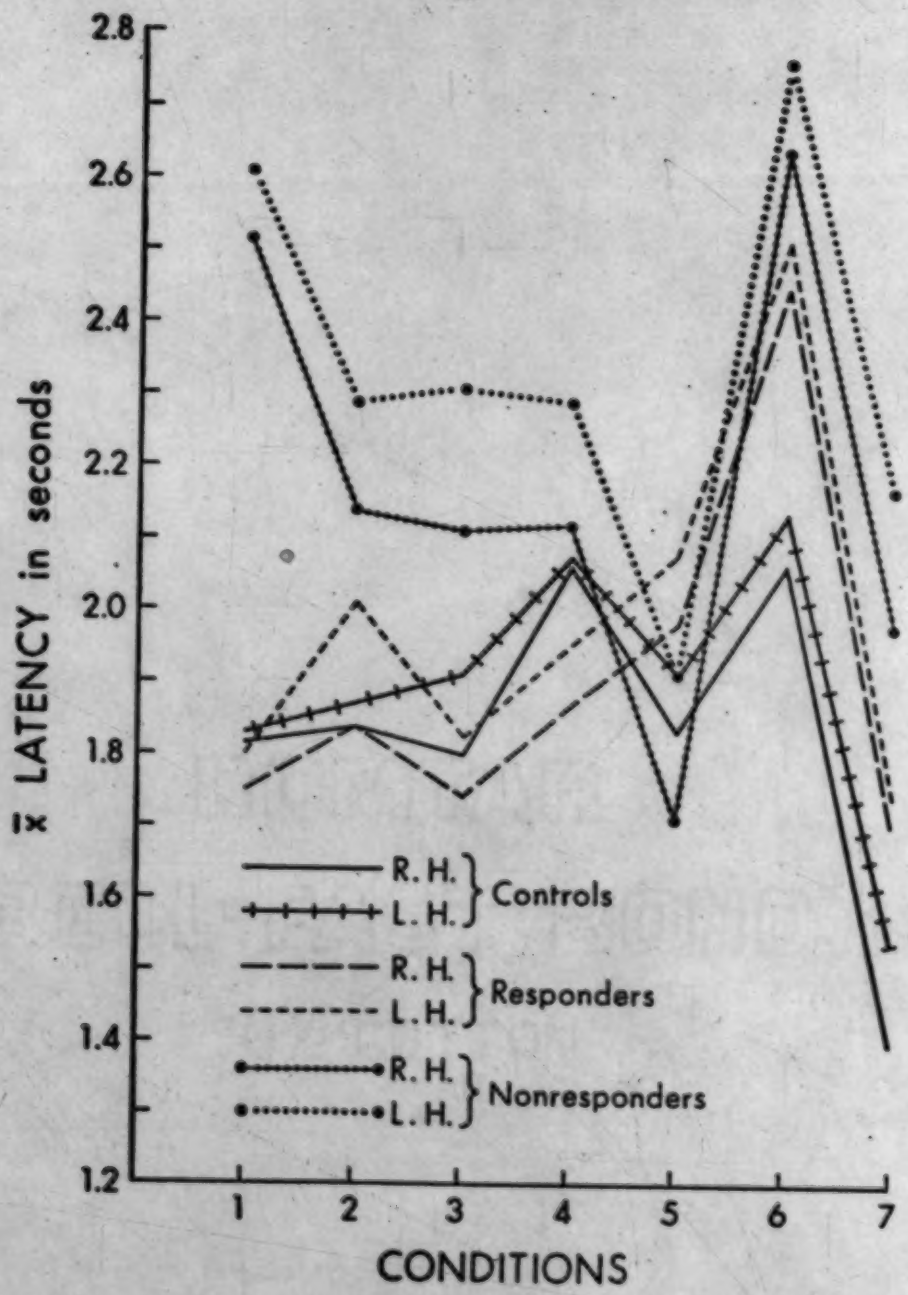


Figure 5. Mean latencies of SCRs for the R and L hands of the three groups during the following: (1) task one, physical stimulation, (2) partial repetition of task one after completion of task two, (3) task eight, startle, (4) task two, habituation/dishabituation, (5) neutral tones and (6) signal tones of task three, (7) task seven, shock.

significantly slower, compared with controls, during task 1 (right hand t (23) = 3.14, $p < .005$; left hand t (23) = 3.52, $p < .001$, 1-tailed comparisons), during task 7 (right hand t (19) = 3.16, $p < .005$; left hand t (19) = 2.46, $p < .01$, 1-tailed comparisons) and compared with responders during task 1 (right hand t (13) = 2.91, $p < .005$; left hand t (13) = 2.67, $p < .01$, 1-tailed comparisons).

Pearson product moment correlations between mean SCR amplitude during cognitive tasks 4 and 5 and the mean latency of task 1 and 7 were nonsignificant for the control group, task 1, right hand r (17) = -.18, $p > .05$; left hand r (17) = -.08, $p > .05$; task 7, right hand r (15) = -.06, $p > .05$, left hand r (15) = -.13, $p > .05$. In the combined schizophrenic groups a relationship between the criterion amplitude measure and latency scores appeared for task 1 but not for task 7; task 1 right hand r (15) = -.50, $p < .06$, left hand r (15) = -.55, $p < .03$; task 7 right hand r (12) = .02, $p > .05$, left hand r (12) = -.27, $p > .05$. However, the correlations on the pooled z scores (task 1, right hand $r_z(15)$ = -.16, $p > .05$, left hand $r_z(15)$ = -.32, $p > .05$; task 7, right hand $r_z(12)$ = -.10, $p > .05$, left hand $r_z(12)$ = -.23, $p > .05$) between the criterion amplitude measure and latency scores for the schizophrenics were nonsignificant. This finding suggests that the correlations between the criterion amplitude measure and latency in task 1 are due to the mean differences in SCR amplitude between the responders and nonresponders rather than a true relationship between latency and the criterion measure.

Changes in latencies within groups across tasks were tested for significance with correlated t tests. The within groups correlated t probabilities indicated that the latency scores changed in a consistent

and significant manner across tasks for each of the three groups (see Tables A, B, C, Appendix B). The responders had shorter latencies compared with controls, on some tasks and longer latencies on others. Two-tailed, independent *t* tests were performed on each task in Figure 5 to test for significance. None of the latencies of the responders differed significantly from those of controls. Other researchers have reported shorter latencies in "responder" schizophrenics compared with controls (Gruzelier & Venables, 1972; Gruzelier, 1973) and shorter latencies in children of schizophrenic mothers with signs of pathology in the children, compared with normal controls (Mednick & Schulsinger, 1968) on simple habituation to repeated tones tasks. In the present study, a similar trend was observed, but it was nonsignificant.

Response latencies were also investigated for bilateral asymmetries. Significant bilateral asymmetries of latencies were found, but for the nonresponder group only. These results are presented in detail in Appendix B.

Response Ascent or Rise Time

The most direct explanation of a skin conductance response is that it is a result of sympathetic activation of the skin area under consideration (Edelberg, 1972a). Although the steepness of the ascent slope appears unrelated to the size of the SCR, when the slope is expressed as a measure of ascent time, this time value is confounded with the SCR amplitude. As a result, meaningful comparisons of slope differences between SCRs of very different amplitudes is not possible. In order to circumvent this problem, ascent slope was also expressed as a ratio of ascent time to SCR amplitude. Thus, small ascent ratios are associated with steep ascent slopes and large ascent ratios are

associated with gentle ascent slopes, regardless of the amplitude of the response.

Figure 6 illustrates the right and left hand ascent time and ratio across tasks for all three groups. Correlated *t* tests within groups indicated that both ascent time and ascent ratio varied significantly across tasks for each group (see *t* score probabilities of Tables A, B, and C in Appendix C).

Independent *t* test comparisons of ascent time for each task between all three groups (Tables D, E, and F, Appendix C) indicated that the ascent times of the nonresponders did not differ significantly, compared with controls, for any of the tasks (Table E, Appendix C). The responders, on the other hand, had significantly shorter ascent times, compared with controls (Table D, Appendix C), and nonresponders (Table F, Appendix C) for several tasks. These shorter ascent times in the responders appeared to occur most frequently during tasks producing large amplitude SCRs in controls and responders. One interpretation of the shorter ascent times in responders is that they are a result of SCRs with similar amplitude values to those of the controls, but with much steeper ascent slopes, and therefore shorter ascent times, than those observed in the controls. A second interpretation is that the ascent slopes are equivalent for the responders and controls, but the peak of the SCR occurs sooner for the responders, thereby resulting in shorter ascent times. In view of the somewhat smaller SCR amplitudes (Figure 3) for the responders during these conditions but equivalent ascent ratios (Figure 6) for the responders and controls, the latter interpretation seems more plausible.

Pearson product moment correlations between the mean criterion

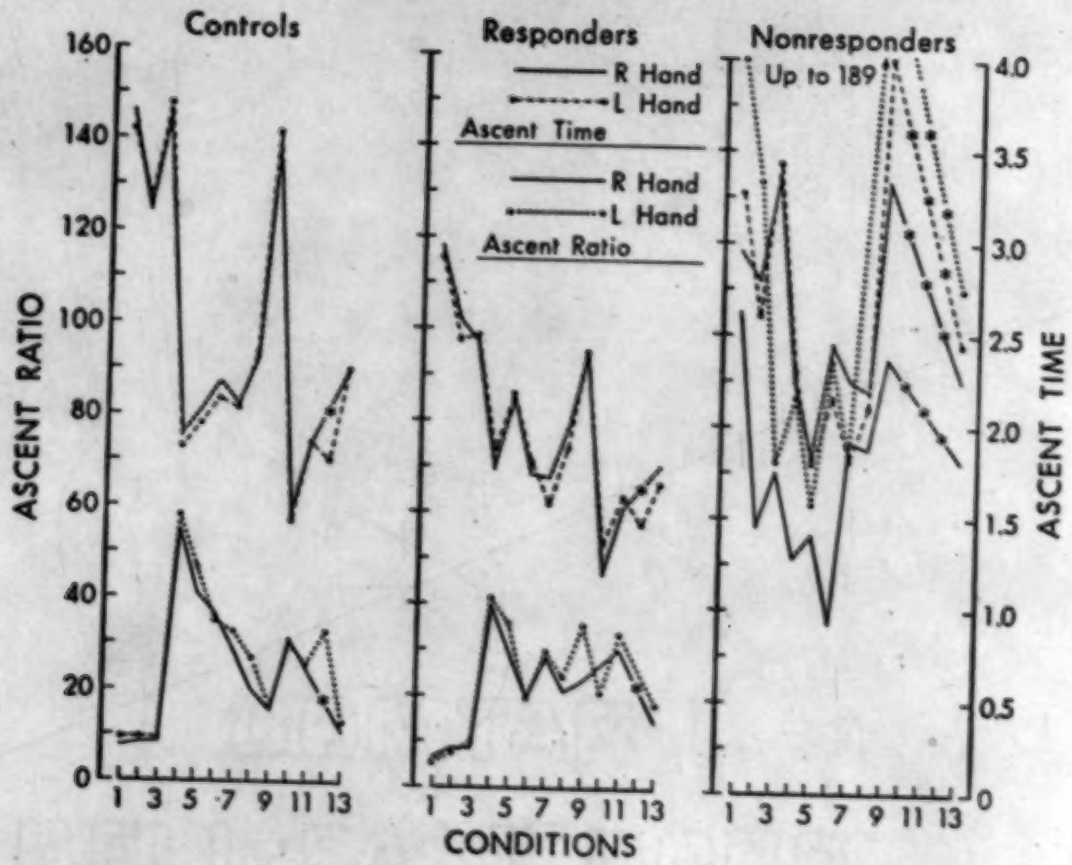


Figure 6. Mean ascent times and ascent ratios of the R and L hands for the three groups during the following: (1) task one, physical stimulation, (2) partial repetition of task one after completion of task two, (3) task eight, startle, (4) task two, habituation/dishabituation, (5) task two, dishabituation alone, (6) spontaneous fluctuations during task two, (7) neutral tones and (8) signal tones during task three, (9) largest amplitude SCRs during instructions and performance of cognitive tasks four and five, (10) SCRs, with recovery times available, during instructions only of cognitive tasks four and five, (11) SCRs, with recovery times available, during performance only of cognitive tasks four and five, (12) SCRs, with recovery times available, of hand not holding pencil during performance of cognitive task six, mirror tracing, and (13) task seven, shock. * on graph indicates lack of data.

amplitude of cognitive tasks 4 and 5 and ascent times and ascent ratios of several tasks are presented in Table 8. Included in Table 8 are the correlations between the criterion variable and ascent time and ascent ratio, after these variables were transformed to within schizophrenic group z scores. The correlations on the raw scores and transformed scores between the criterion amplitude and ascent time are nonsignificant for both the control and combined schizophrenic groups. The correlations on the raw scores between the mean SCR criterion amplitude measure and ascent ratios are negative and significant for several tasks in the control and combined schizophrenic groups, with trends in the same direction on the other tasks. The correlations on the transformed scores also indicate an inverse relationship between the SCR criterion amplitude values and the ascent ratio values, but not as consistently as the raw score correlations.

For the nonresponder group, the ascent ratio was significantly different from that of the control (Table E, Appendix C) and that of the responder group (Table F, Appendix C) for several tasks illustrated in Figure 6. This result appears to be due to the inverse relationship between ascent ratio and the amplitude of the criterion SCR observed in both the control and combined schizophrenic groups (Table 8).

Response Recovery Time

Data presented by Edelberg (1970) on the correlation between SCR amplitude and one-half amplitude recovery time suggests no consistent relationship between these two measures. However, as was true of ascent time, the time expression of SCR recovery is confounded with amplitude and does not allow for meaningful comparisons of SCR recovery between responses of very different amplitudes. Therefore, the recovery time

TABLE 8

Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean ascent times and mean ascent ratios during several tasks. r_z = product moment correlations on these same variables after the within schizophrenic group raw scores were converted to z scores and then pooled for the correlations. Upper correlation is with ascent time, lower correlation is with ascent ratio.

Task Number & Description	Normals				Schizophrenics					
	Right Hand		Left Hand		Right Hand			Left Hand		
	N	r	N	r	N	r	r_z	N	r	r_z
1 Physical Stimulation	17	-.11	17	-.15	15	-.23	-.48	15	-.20	-.13
	17	-.31	17	-.41	15	-.41	-.33	15	-.44	-.08
1 Partial repeat after 2	16	-.38	16	-.15	11	-.09	.14	11	0.0	.25
	16	-.004	16	-.12	11	-.66*	-.43	11	-.66*	-.38
4, 5, largest amplitude SCR during arithmetic and spelling backwards	17	.25	17	.36	12	-.51	-.09	12	-.48	-.01
	17	-.71***	17	-.75***	12	-.70**	-.34	12	-.67*	-.63*
4, 5, 6, SCRs, with available recovery time	17	.15	17	-.01	10	-.11	-.78**	10	-.19	.31
	17	-.40	17	-.57*	10	-.28	-.22	10	-.48	-.11
7 Shock	15	-.26	15	-.28	12	-.23	.27	12	-.29	.30
	15	-.52*	15	-.56*	12	-.36	-.42	12	-.42	-.49
8 Startle	17	-.11	17	-.11	14	-.20	.11	14	-.20	.19
	17	-.55*	17	-.58*	14	-.50	-.44	14	-.49	-.58*

$p < .05$*

$p < .01$**

$p < .001$***

measures in the present study were also expressed as ratios by dividing the one-half amplitude recovery time by one-half the amplitude. Very steep recovery slopes resulted in small ratios and gentle recovery slopes resulted in large ratios.

The right and left hand recovery times for all three groups during 14 situations are shown in Figure 7a and the corresponding recovery ratios are shown in Figure 7b. For all three groups the recovery time of the electrodermal response generally became shorter as the attentional value of the stimulus increased (see *t* score probabilities across tasks in Tables A, B, C, Appendix D), thus supporting the results of Edelberg (1972b). The SCRs, with available recovery times, during cognitive tasks 4, 5, and 6 had the shortest recovery times.

The results of the present study cast some doubt on the conclusion by Edelberg (1972b) that defensive responses such as threat of shock resulted in longer recovery times than those associated with "goal-oriented" tasks. Although the recovery time data supports Edelberg's conclusion the recovery ratio data do not. Edelberg did not report what the SCR amplitude differences were between his tasks. Unless the amplitudes were equivalent, it is likely that Edelberg's data was confounded by SCR amplitude differences.

No significant differences in recovery times were observed in the responders, compared with controls (independent *t* test Table D, Appendix D). However, the most consistent differences in recovery times between these two groups which did appear (Figure 7a, situations 4 and 5) occurred during the repeated presentation of tones task (number 2) where the responder recovery times were shorter. Gruzelier and Venables (1972) and others (Ax & Bamford, 1971) have all reported shorter recovery

Figure 7a. Mean one half amplitude recovery times of the R and L hands for the three groups during the following: (1) task one, physical stimulation, (2) partial repetition of task one after completion of task two, (3) task eight, startle, (4) task two, habituation/dishabituation, (5) task two, dishabituation alone, (6) spontaneous fluctuation during task two, (7) neutral tones and (8) signal tones of task three. SCRs, with recovery time available, during (9) instructions, (10) 30 second base period, and (11) performance of cognitive tasks four and five. SCRs, with recovery times available, during (12) instruction, and (13) performance of cognitive task six. (14) task seven, shock. * on graph indicates lack of data.

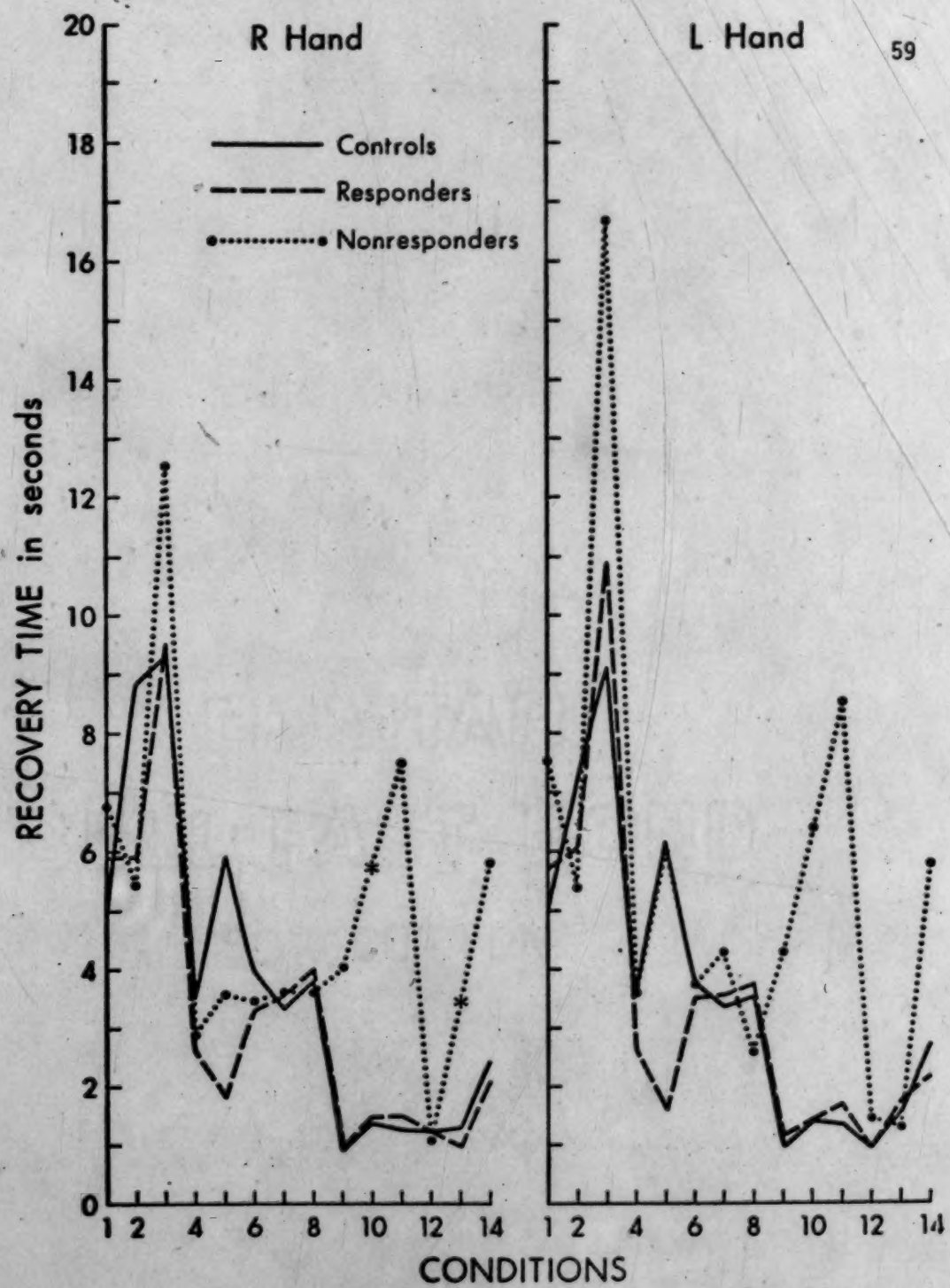
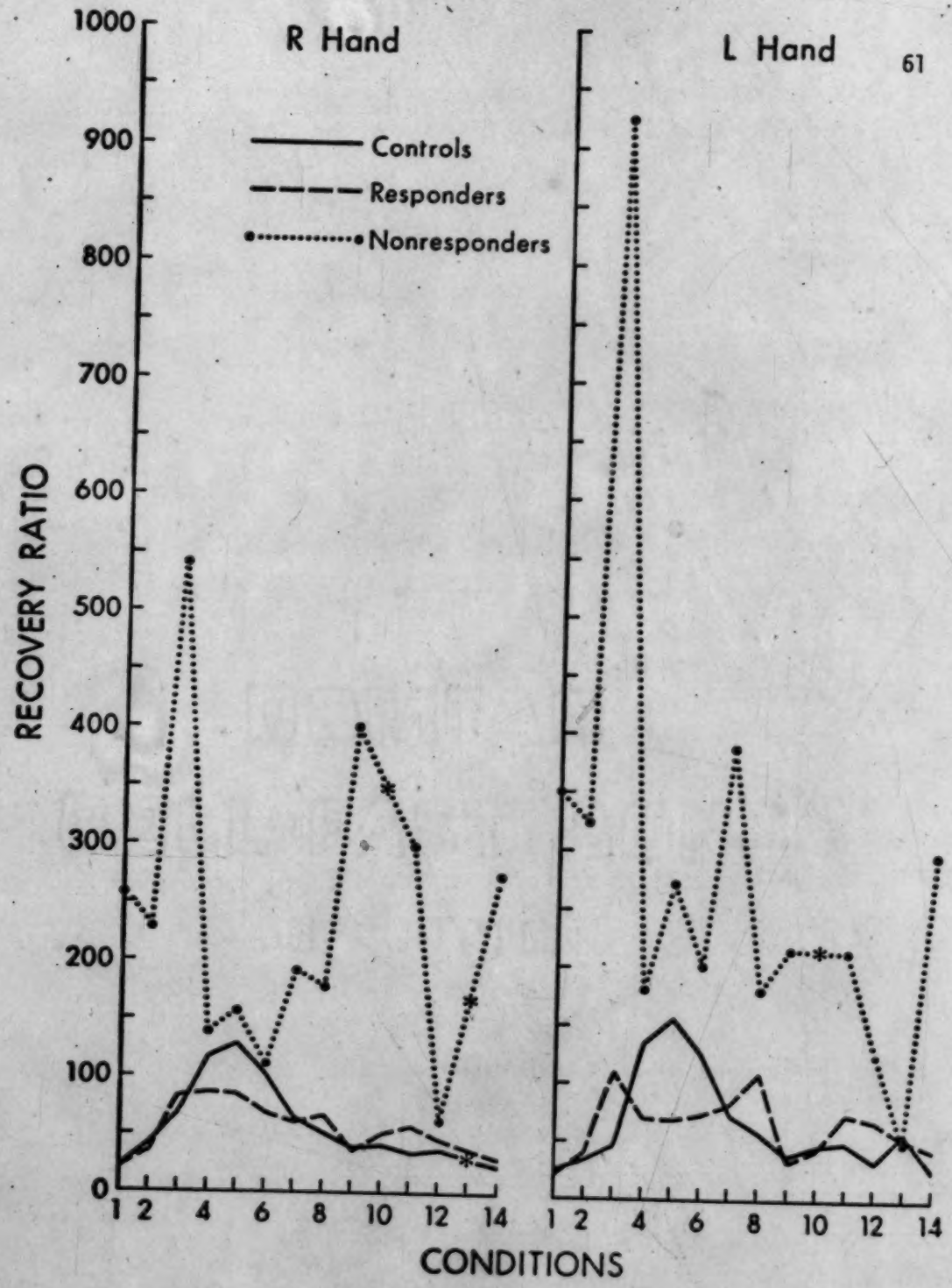

R Hand
L Hand
Controls
Responders
Nonresponders
RECOVERY TIME in seconds
0
2
4
6
8
10
12
14
16
18
20
1 2 4 6 8 10 12 14
1 2 4 6 8 10 12 14
CONDITIONS

Figure 7b. Mean one half amplitude recovery ratio of the R and L hands for the three groups during the following: (1) task one, physical stimulation, (2) partial repetition of task one after completion of task two, (3) task eight, startle, (4) task two, habituation/dishabituation, (5) task two, dishabituation alone, (6) spontaneous fluctuation during task two, (7) neutral tones and (8) signal tones of task three. SCRs, with recovery time available, during (9) instructions, (10) 30 second base period, and (11) performance of cognitive tasks four and five. SCRs, with recovery times available, during (12) instruction, and (13) performance of cognitive task six. (14) task seven, shock. * on graph indicates lack of data.


R Hand
L Hand
Controls
Responders
Nonresponders
RECOVERY RATIO
1000
900
800
700
600
500
400
300
200
100
0
1 2 4 6 8 10 12 14
1 2 4 6 8 10 12 14
CONDITIONS

times of SCRs to repeated tones in some schizophrenics compared with controls.

The recovery times of the nonresponding schizophrenics were also very similar to those of the control and responder groups (Tables E, F, Appendix D) and differed significantly for only one task. Shock delivery (task 7) resulted in significantly longer recovery times for the nonresponders, compared with controls (right hand t (18) = 4.4, $p < .001$; left hand t (17) = 4.0, $p < .001$) and with responders (right hand t (10) = 3.2, $p < .01$; left hand t (9) = 5.5, $p < .001$).

Pearson product moment correlations between the mean SCR criterion amplitude of cognitive tasks 4 and 5 and the recovery times and recovery ratios of several tasks are presented in Table 9. For the schizophrenic group, correlations are also presented in Table 9 for these same variables after the variables were transformed to within group z scores. Correlations, for the raw and transformed scores, between the criterion amplitude and recovery time are nonsignificant for both the control and combined schizophrenic groups, thus providing no support for a relationship between recovery time and the mean SCR amplitude on the criterion tasks.

The recovery ratios of the nonresponders (Figure 7b) were significantly larger compared with those of controls, for all tasks with recovery data available except task 2 and the spontaneous fluctuations (see independent t tests, Table E, Appendix D), and, to a lesser extent, significantly larger than those of the responders (Table F, Appendix D). That is, the recovery slopes of the nonresponder SCRs were more gradual than those of the controls and responders for most of the SCRs. The correlations between the SCR amplitude criterion measure and recovery

TABLE 9

Pearson product moment correlations between criterion amplitude for categorizing schizophrenics and mean recovery times and mean recovery ratios. r_z = product moment correlations on these same variables after the within schizophrenic group raw scores were converted to z scores and then pooled for the correlations. Upper correlation is with recovery time, lower correlation is with recovery ratio.

Task Number & Description	Controls				Schizophrenics					
	Right Hand		Left Hand		Right Hand			Left Hand		
	N	r	N	r	N	r	r_z	N	r	r_z
1 Physical Stimulation	16	.002	16	.10	13	-.01	0.0	13	-.27	-.03
	16	-.26	16	-.26	13	-.41	0.0	13	-.49	-.23
2 Habituation/Dishabituation	16	-.10	16	.04	14	.27	.27	13	-.11	.31
	16	-.35	16	-.18	14	-.12	.22	13	-.45	-.09
4, 5, and 6, SCRs, with recovery time available	17	-.24	17	-.26	9	-.12	.57	10	-.21	.15
	17	-.52*	17	-.51*	9	-.42	-.22	10	-.42	-.03
7 Shock	14	-.27	14	-.33	12	-.10	.02	11	-.56	.08
	14	-.44	14	-.49	12	-.49	-.40	11	-.50	-.54
8 Startle	14	-.43	14	-.25	7	-.22	-.08	8	-.21	-.10
	14	.02	14	-.60*	7	-.41	-.56	8	-.36	-.50

$p < .05$*

$p < .01$**

$p < .001$***

ratios in the normal controls (Table 9) support the presence of an inverse relationship between the criterion variable and recovery ratios and would thus appear to account for the data. However, much less support for this inverse relationship exists in the combined schizophrenic group correlations for both the raw and transformed scores.

Skin Conductance Levels

Of interest in the present study were mean skin conductance level differences between groups. Gruzelier and Venables (1972) reported SCLs significantly above controls for responders and SCLs significantly below controls in nonresponders. Mean SCLs for the right and left hands of all three groups for seven situations are illustrated in Figure 8. The mean SCL of the control group was highest, followed by those of the responding and nonresponding schizophrenic groups, respectively. Two way repeated measures analyses of variance were performed on the right and left hand level scores. Differences in mean SCLs between groups were significant, right hand F (2,29) = 8.6, $p < .001$; left hand F (2,29) = 10.1, $p < .001$. The lower SCLs in responders, compared with controls, were an unexpected result. Although it is possible to argue that the heavy medication regime of the schizophrenics accounts for their low SCLs (Tecce & Cole, 1972), even this argument has little support in the rank order correlations of medication levels with SCLs (Table 3 above).

Pearson product moment correlations between the mean SCR criterion amplitude of tasks 4 and 5 and the mean SCL over the entire session were nonsignificant for the control group, right hand r (17) = -.09, $p > .05$; left hand r (17) = .17, $p > .05$. In the combined schizophrenic group, however, the correlations suggest a relationship between the SCR criterion amplitude measure and the SCL, but only for the left hand;

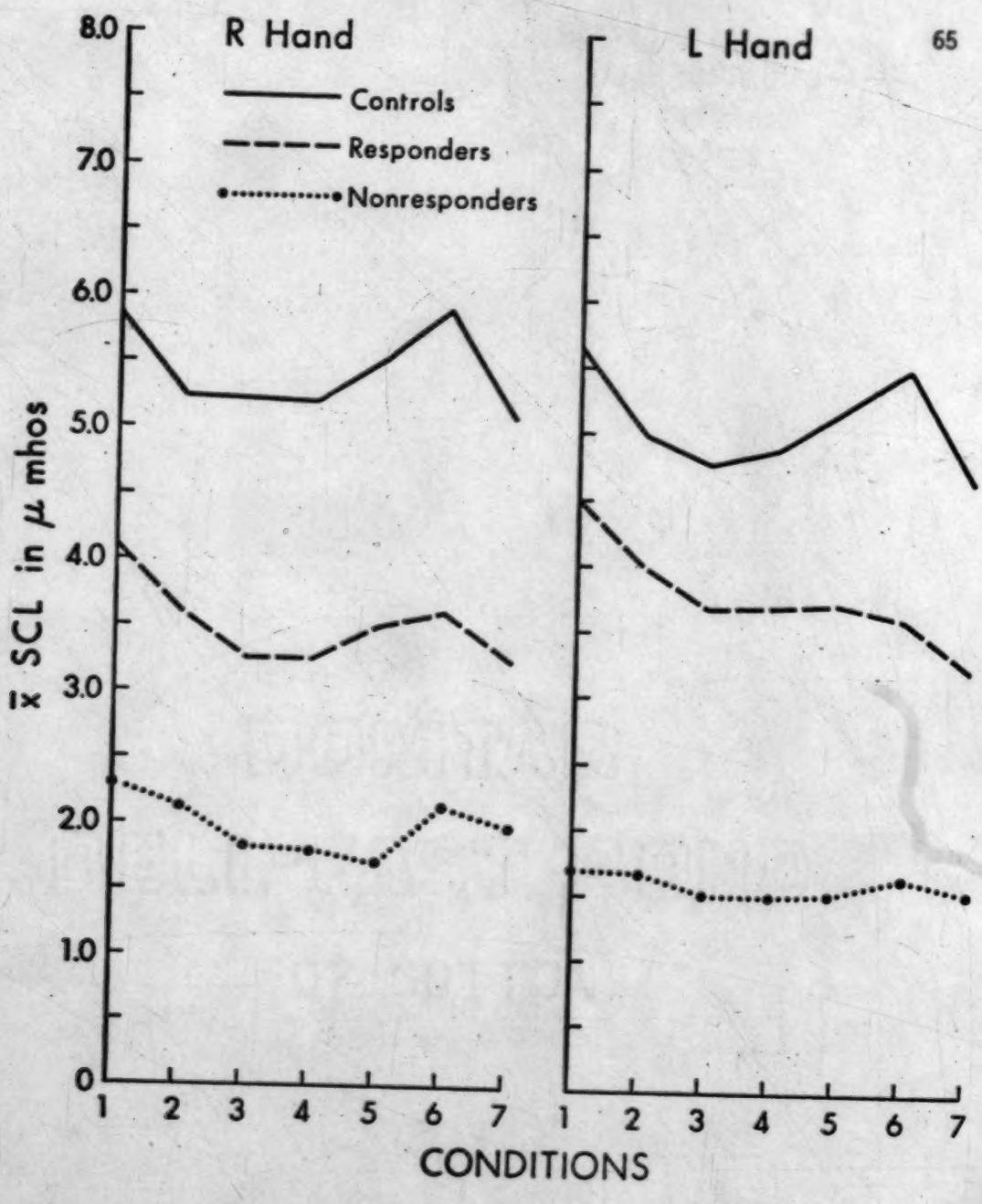


Figure 8. Mean R and L hand SC levels for the three groups determined just prior to the SCRs for the following situations: (1) task one, physical stimulation, (2) task two, habituation/dishabituation, (3) neutral tones and (4) signal tones of task three, (5) during performance of cognitive tasks four and five, (6) task seven, shock, and (7) task eight, startle.

right hand r (15) = .40, $p > .05$; left hand r (15) = .65, $p < .01$; right hand r_z (15) = .27, $p > .05$; left hand r_z (15) = .46, $p > .05$.

Detailed analyses of the SCL changes during cognitive tasks were carried out in the present study. Lacey, Bateman, and VanLehn (1953) reported increases over a resting baseline in SCL during instructions, during a one minute baseline period following instructions, and during the actual performance of the cognitive problems. Although these authors do not state precisely how they measured these levels, they appeared to select the highest SCLs attained within the designated time periods. Rather than using the highest SCLs attained, which is difficult to separate from the aftermath of an SCR, in the present study a crude average of SCL within each time period was used to obtain a more conservative estimate of SCL change.

Each subject pre-instruction SCL was subtracted from the mean SCLs during the instruction, intermediary base period and performance of the task and these change scores were analyzed. These three mean difference scores, one set for each hand, are shown in Figure 9 for each of the groups. This graph shows that the average change in SCL for the normal group was always an increase and that the SCL changed very little for the nonresponder schizophrenic group. Correlated t scores were calculated on each difference score to determine if the average change within each group was significant. For the control group the SCL changes were significant during instructions (right hand t (16) = 5.36, $p < .01$, left hand t (16) = 5.63, $p < .01$) and during problems (right hand t (16) = 3.29, $p < .01$; left hand t (16) = 3.72, $p < .01$) and during the base period the left hand changes were significant (t (16) = 2.34, $p < .05$). None of the SCL changes for either

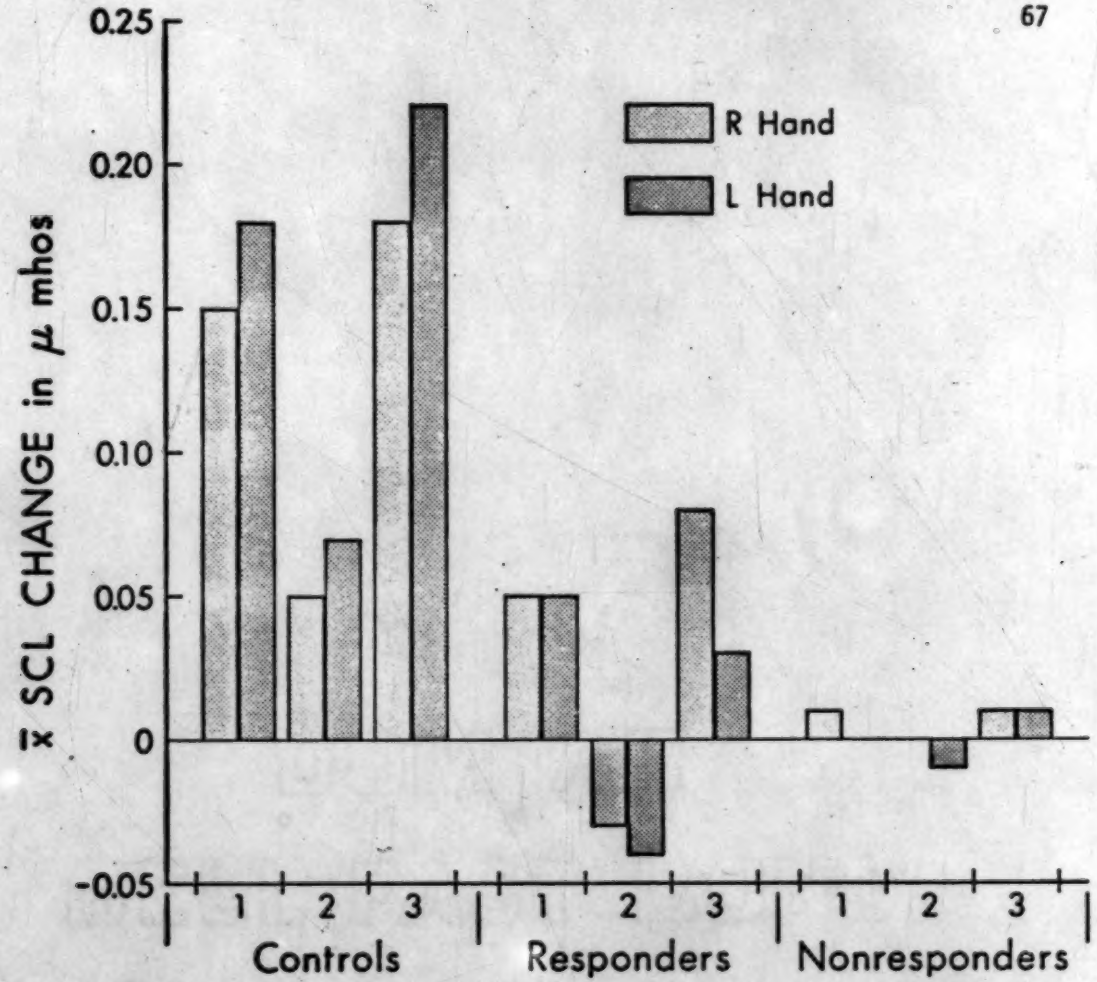


Figure 9. Mean SCL change scores during cognitive tasks four, arithmetic, and five, spelling backwards, for the three groups. The SCL prior to the cognitive tasks is subtracted from each SCL measured during the tasks. (1) Change scores during instructions to tasks four and five. (2) Change scores during rest period after instructions. (3) Change scores during performance of tasks.

of the schizophrenic groups approached significance. The increases in the control group levels supports the findings reported by Lacey et al. (1953), but these effects were not replicated in the two schizophrenic groups.

The presence of bilateral asymmetrical differences in SCLs was also of interest in the present study. Gruzelier and Venables (1973) reported higher SCLs of the right hand in their responder group and higher SCLs of the left hand in their nonresponder group and no consistent differences in SCLs for the control group. These results were not replicated in the present study and are presented in detail in Appendix E.

Heart Rate Response

The most significant effect found on examination of the heart rate data was a consistent elevation of the basal heart rate of all subjects on medication, a finding consistent with results reported by Tecce and Cole (1972). The basal heart rate increases were so consistent that the two unmedicated schizophrenics had data which could be sorted out from that of the medicated schizophrenic data on the basis of the slower HR alone. In Table 10, the mean basal HR before the habituation task and at the end of the first 15 tones is presented for the three experimental groups. The report of significantly higher heart rates in responders compared with nonresponders (Gruzelier & Venables, 1975) was not supported by the present study.

The degree of acceleration or HR change score in response to breath inhalation and startle stimuli was less for the medicated schizophrenics grouped together, when compared with normal controls. The baseline minus the acceleration score (B - H:HR, see methods section) for the

TABLE 10

Basal Mean HR before and after Habituation

Subject	N	Before	After
Unmedicated Schizophrenics	2	65.9	65.6
Normal Controls	17	70.6	70.6
Medicated Responders	6	90.9	92.3
Total Responders	7	87.0	87.0
Medicated Nonresponders	7	98.4	92.3
Total Nonresponders	8	93.8	88.2

control group was .21 seconds, whereas it was only .11 seconds for the medicated schizophrenics. This finding is consistent with the reports of others such as Cannon (1932) who suggested the operation of homeostatic mechanisms of restraint and Wilder who postulated a statistical expression of restraints (Wilder's Law of Initial Values; Wilder, 1958).

Since the responder and nonresponder groups differed greatly on electrodermal activity measures evidence was sought for differences in HR responsiveness between these two groups. Mean change scores were computed for each subject on the slowest (L: HR) minus the fastest (H: HR) interbeat interval values (L - H: HR) for tasks 1, 7, and 8. No difference was found. The mean values of the L - H: HR scores was .27 seconds for the responder group and .25 seconds for the nonresponder schizophrenics.

During the habituation/dishabituation task the schizophrenic subjects and the control subjects showed accelerative and decelerative components of HR change to the tones. However, in contrast to the report by Holloway and Parsons (1971) that the accelerative component (H: HR) habituated over trials, no such habituation occurred for any of the three groups in the present study. These results were unexpected. In order to assure that mean values across subjects were not averaging out individual change scores, a difference score analysis of the control group data was undertaken on the accelerative component. The mean base prior to the presentation of tones (BL: HR) for each of the control subjects was averaged into the subject's grand mean ($\bar{X}$ BL: HR) and the accelerative component of each tone presentation was subtracted from this grand mean ($\bar{X}$ BL - H: HR). It was evident from the pattern of change scores that the fastest components over the 18 trials were

simply not occurring at the start of the tones presentation or during the novel tones at the end of the habituation period. The fastest changes appeared to occur at a different point for practically every subject. The possibility that random changes in respiration were confounded with accelerative change scores due to the presentation of tones was investigated. Respiration changes could not account for the data although the random changes which did occur at approximately the same time as a tone were obviously adding "noise" to the HR change scores. Holloway and Parsons (1971) did not control for respiration changes.

The control group data were examined for differences in HR change between the signal tones requiring a motor response and the neutral tones which required no response from the subject. It was assumed that the decelerative component would be reduced during the motor response (Obrist, Webb, & Sutterer, 1969). Neither the fast nor the slow components of the HR response appeared to differ. The mean of the fastest IBIs (H: HR) was .70 seconds for the neutral tone and .67 seconds for the signal tone; the mean of the slowest IBIs (L: HR) was 1.03 for the neutral tone and 1.02 for the signal tone.

Respiration Rate Response

Respiration rate has been used as an index of autonomic activity by a number of researchers (Pinneo, 1961; Reynolds, 1962; Williams, 1953). Higher respiration rates have been reported in schizophrenics compared with rates in normals (Gunderson, 1953; Jurko, Jost, & Hill, 1952; Williams, 1953). Since phenothiazines do not alter respiration rates (McDonald & Gynther, 1962) it appears to be a useful index in the present study to determine differences in arousal levels for the

two schizophrenic groups. The HR data tends to suggest equal arousal levels for the two schizophrenic groups since the elevation of HR by medication did not differ between the two groups.

Number of inspirations per minute were determined at the beginning, midpoint, and end of the tone habituation/dishabituation series and during the relaxed period at the end of the session. A mean for each subject was obtained (see Table 11).

The mean respiration rates of the two schizophrenic groups were significantly higher than those of the control group.

Neuropsychological Data

The neuropsychological variables, completed for 13 normals, seven responders, and six nonresponders, are summarized in Table A of Appendix F. A few of the variables are occassionally short of one subject due to the lack of cooperation on some of the tests by that subject. The WAIS scores are presented for the schizophrenics only (Table B, Appendix F) since so few of the controls (N = 5) were administered the test. In some cases the subjects were reticent to take the test; others were too test wise to provide valid data.

From the tables in Appendix F it is evident that the nonresponders had the lowest mean IQ (76.3) and that they showed significantly greater deficits on the neuropsychological test variables compared with the deficits of the responding schizophrenic group. Of 32 variables listed in Table A of Appendix F the nonresponders were significantly more impaired (Scheffe comparisons, $p < .05$) than the normals on 27 of the variables and significantly more impaired than the responders on ten variables. In contrast, the responders were significantly more impaired than the controls on only eight variables and had a higher

TABLE 11

Mean Inspiration per minute for each group. (Three samples during Habituation/Dishabituation Task and one at the end of the test session).

N	Group	$\bar{X}$	Mann-Whitney U (one-tailed test)
17	Normal Controls	14.0	Normals/Responders $U = 8$, $p < .001$
7	Responders	20.5	Normals/Nonresponders $U = 38$, $p < .05$
8	Nonresponders	17.2	Responders/Nonresponders $U = 14$, $p < .06$

mean IQ (91.7) than did the nonresponders.

A stepwise discriminant function analysis of the neuropsychological variables alone yielded 100% correct classifications for all three groups.

DISCUSSION

Significant Differences: Group Attributes or Statistical Artifacts?

The general conclusion to be drawn from this study is that schizophrenics can be divided into two distinctly different populations on the basis of their electrodermal reactivity. On a number of separate electrodermal response measures responder and nonresponder schizophrenics differ from each other and the control population in many significant ways.

However, certain questions arise in regard to the methodology used in the present study. The division of the schizophrenic group into responders and nonresponders does not meet the assumption that these two groups represent random samples from two distinct populations. Rather, the groups may simply represent the extremes of one continuous population. Therefore, the division of the two schizophrenic groups based on one criterion measure of electrodermal activity could lead to significant group differences on other electrodermal measures which in turn would be entirely the result of the correlation of the criterion variable with other electrodermal measures investigated in the study.

Although Gruzelier and Venables (1972) have not addressed themselves to this methodological problem, the present findings of this study indicate that the significant differences found between the two schizophrenic groups represent separate characteristics of these groups, and are not the result of relationships between the electrodermal measures and the criterion measure utilized to differentiate the schizophrenics into responders and nonresponders.

Pearson product moment correlation coefficients were computed between the mean SCR amplitude during the criterion task for differentiating

schizophrenics into two groups (tasks 4 and 5) and other electrodermal response measures which yielded significant differences between groups and were reported in the results section in the presentation of findings pertinent to that particular measure. In some instances additional events which had failed to yield significant differences between groups on that variable had correlations calculated for comparison purposes. Since a significant correlation between two variables in the combined schizophrenic group could be the result of (1) a true relationship between these two variables, or (2) an artificial effect due simply to the differences between the means of these groups on a variable, a second Pearson product moment correlation for the combined schizophrenic groups was performed. This second correlation was computed after the subjects' scores within a group were standardized into z scores within that group and the z scores were pooled from both groups.

In the correlations between the criterion SCR amplitude and a subject's SCR amplitude on other tasks, evidence of significant correlations in both the control group and combined schizophrenic groups suggests a relationship between these variables on several tasks. Furthermore, the significant correlations on the z scores in the combined schizophrenic groups did not disappear.

Frequency counts of SCR responses during the habituation/dishabituation task, the tones attentional task and the number of spontaneous fluctuations did not appear to be related to the criterion amplitude of the SCRs during cognitive tasks for the control group. For the combined schizophrenic groups, the correlations on raw and transformed scores were consistent and suggested a relationship between the SCR criterion variable and frequency of responding on task 3 (tones

attentional task) for the left hand only.

The correlations between the criterion variable and latency on independent tasks for the controls were insignificant, suggesting an orthogonal relationship between latency of SCRs and SCR arousability, as measured by the criterion task. In the combined schizophrenic groups, some correlations on the raw scores were significant; however, these significant effects were not evident in the correlations on the transformed scores. Therefore the relationship between the SCR criterion variable and SCR latency in the schizophrenics also appears to be orthogonal.

Correlations on the raw scores for both the controls and schizophrenics and on the z scores for the schizophrenics between amplitude of SCRs on the criterion tasks and ascent time and recovery time on several tasks were all nonsignificant. This result suggests that the mechanism determining the width of the "ascent windows" and "recovery windows" acts independently of the SCR amplitude attained within these "windows".

Significant inverse correlations between the criterion SCR amplitude variable and the ratio expression of ascending slopes in both the control and combined schizophrenic groups support an interpretation of a strong relationship of ratio expressions of ascent slope with the subject's ability to respond with large amplitude SCRs. For the control group, an inverse relationship between ratio expressions of recovery slopes and the subject's ability to respond with large amplitude SCRs also appears to exist. For the combined schizophrenic groups however, neither the raw score correlations nor the z score correlations are significant or consistent. The absence of a relationship between the ratio expression of recovery slopes and the SCR criterion measure in the

combined schizophrenic groups may be due to lack of within group consistency between these two variables for the two schizophrenic groups.

The correlations between SC levels and SCR amplitude of the criterion response were very low and insignificant in the control group, suggesting an orthogonal relationship between these two variables. For the combined schizophrenic groups, the raw and z score correlations support a relationship between the criterion variable and the SC level of the left hand only. The consistency with which a relationship appears between the criterion variable and amplitude of SCRs on other tasks, frequency of responding, and SC level does not suggest that these are random effects. However, this author has no reasonable explanation for this phenomena.

It is reasonable to suppose that the duration of hospitalization represents a behavioral assessment reflecting the judgments of psychiatrists, among other things, with respect to the progress of the patient's illness. Nonresponders had significantly longer durations of hospitalization and were more often considered to be "chronic" patients. The relationship between the continuum of acute and chronic and the dimension of responder-nonresponder is not a simple one-to-one association; however, it is better viewed in terms of degree of overlap. Re-examination of hospitalization records at the conclusion of the present study revealed that, regardless of the criteria of chronicity employed, sorting of the schizophrenics via their hospitalization record would have resulted in a mixture of responders and nonresponders in both acute and chronic categories.

That the nonresponders are significantly different from the responders and from controls as well is evident in the neuropsychological

data. The nonresponders were significantly more impaired on the neuropsychological and WAIS assessments than were the responders. The mean level of performance of the nonresponders was worse than that of the controls and responders on all but two of the 32 neuropsychological variables. The responder's performance fell between that of the controls and nonresponders on all tests except Wepman-Jones Aphasia test and the Organiz Integrity test where the responder's performance fell slightly below that of the nonresponders. This consistency in performance across most of the neuropsychological variables no doubt accounts for the 100% correct classifications of all of the subjects on the basis of a stepwise discriminant function analysis. Such complete separation of the schizophrenic population on the basis of their performance on the neuropsychological test battery and also on the basis of their categorization by an independent criterion variable, electrodermal responsivity, strongly suggests that the differences on some of the electrodermal measures between these two groups reflect different underlying characteristics of these groups and the population from which they were drawn.

A possible criticism of the statistical analysis in the present study was the use of univariate rather than multivariate analysis (see Appendix H).

Similarities and Differences between the Present and Other Studies

In spite of methodological, task, medication level, and other differences between the present study and those of Gruzelier and Venables, a number of similar findings can be reported and these substantiate the literature which suggests a breakdown of the schizophrenic population into two groups on the basis of electrodermal activity.

The higher number of spontaneous fluctuations in responders and lower number of spontaneous fluctuations in nonresponders compared with

controls, is consistent with the data reported by Gruzelier and Venables (1972) and Gruzelier (1973).

The significantly faster SCR latencies of responders during the repeated presentations of tones task compared with controls (Gruzelier, 1973; Gruzelier & Venables, 1973) was not found in the present study; however, the data on latency curves (figure 5) indicates a trend towards faster latencies on this task and on simple tasks such as breath inhalation and startle stimulus for the responder group. In a similar tones habituation task, Mednick and Schlusinger (1968) reported faster latencies in high risk children who eventually showed signs of psychopathology.

Gruzelier and Venables reported longer latency for nonresponders during signal tones, compared with responders although not significantly so. A comparison with a normal control group was not available in their study (Gruzelier & Venables, 1973; Gruzelier, 1973). A similar trend was observed in the nonresponders during the signal task in the present study when they were compared with both responders and controls, but it also was not significant. However, during task 1 (breath and cough stimuli) and task 7 (shock presentation) the latencies of the nonresponders were significantly slower than those of the other two groups in the present study.

Several researchers have reported faster SCR recovery measures of responding schizophrenics during simple presentations of tones, compared with controls (Ax & Bamford, 1971; Gruzelier & Venables, 1972). In the present study both recovery time and recovery ratio measures indicate a trend of consistently faster SCR recovery rates for responding schizophrenics compared with controls, but only for the repeated presentations of tones (task 2) (see Figure 7a, b, and Table A, Appendix D).

The recovery ratio data (Figure 7b) in the present study shows that short recovery times occurred during goal-oriented tasks for controls and responders and that recovery times were always shorter for responders than for normals in response to simple tones and during spontaneous fluctuations. Since Gruzelier and Venables (1972) reported larger amplitude SCRs in their responding schizophrenics during repeated presentations of tones compared with controls, the significantly shorter recovery times reported by them would have been enhanced had they controlled for amplitude effects in the manner employed in the present study.

Certain differences were found between the results of the present study and those of Gruzelier and Venables which may result from the use of a different criterion measure to differentiate the responders from nonresponders, varying attentional demands or other aspects of the tasks, a considerable difference in medication levels, or differences in the schizophrenic populations.

The criterion measure used in the present study differed from those employed in other studies and may be responsible for differences in the nature of the responder and nonresponder groups in the present study compared with those described by Gruzelier and Venables (1972). While these authors (Gruzelier & Venables, 1972) have reported that responders and nonresponders occurred with equal frequency in acute and chronic populations of schizophrenics,in the present study responders and nonresponders were found with equal frequency in the schizophrenic population but differed significantly in chronicity, the nonresponders having the longer history of hospitalization.

It also appears that the present study employed criterion tasks

which possessed much more atentional value than did those used by Gruzelier and Venables. The more obvious differentiation of attentional demands may have been necessary in light of the heavier medication levels of the experimental subjects in the present study. It is plausible to suppose that the responders in the present study if given less medication would have more closely resembled those in the studies of Gruzelier and Venables to the extent of showing less habituation of SCRs to tones and higher SCLs compared with controls (Tecce & Cole, 1972). It is also plausible that the nonresponders, with less medication would have responded electrodermally to the less demanding attentional task of differentiating signal from neutral stimuli with a motor response (pressing a button) as was reported by Gruzelier and Venables (1972).

However, these speculations cannot account for the initial presence of SCRs to repeated tones, a task of low attentional demand, which occurred in 50% of the nonresponders, or the absence of SCR reactivity to both signal and neutral tones, in a task with more attentional demands than has the habituation task, in the nonresponder group. It appears that factors other than the differences in tasks, medication levels, and methods of group selection are necessary to account for the differences in results. A suggestion by Depue and Fowles (1976), discussed in more detail later, that one group of schizophrenics respond only during nonstressful experimental procedures and decrease their electrodermal reactivity with increases in stressful experimental procedures while another group of schizophrenics increase their electrodermal reactivity with increases in stress, much like controls do, would account for the results reported in the present study but not for the results of the nonresponders in the studies by Gruzelier and

Venables. These authors (Gruzelier & Venables, 1973) reported that nonresponders increased their reactivity when faced with a more stressful task (pressing a button to signal but not to a neutral tone). In spite of these contradictory results it is still plausible that with even greater increases in the stress of the tasks employed, Gruzelier and Venables would have also found less SCR amplitude increases, possibly even decreases in the reactivity of their nonresponders.

Reports (Gruzelier & Venables, 1972; Gruzelier, 1973) of higher SCLs in responding schizophrenics and lower SCLs in nonresponders compared with controls were not replicated in the present study. Responders had SCLs below those of the control group which may reflect the heavier medication regimes of the present study, although this interpretation is not strongly supported by the rank order correlations between medication regimes and SCLs, which were very low.

Gruzelier and Venables reported significant bilateral differences in SCLs and in response amplitudes for certain tasks and cautiously interpreted the findings in terms of unilateral dysfunction of the temporal lobe ipsilateral to the side of the dysfunction (Gruzelier, 1973; Gruzelier & Venables, 1973). Flor-Henry has also hypothesized a unilateral dysfunction in schizophrenics lateralized to the dominant lobe. Consistent significant bilateral SCL and amplitude differences were not observed in the present study. There is evidence that the heavy medication regime of the present study's subjects may have masked bilateral asymmetries of SCLs and/or amplitudes since, in an ongoing study, Gruzelier (personal communication, 1975) has observed that an enhancement of SCL asymmetries occurred during a period of four weeks when medication was withdrawn from subjects and these asymmetries were

attenuated with the resumption of medication. However, since the average nonsignificant differences in SCLs were in the opposite direction from those reported by Gruzelier and Venables (1973) for both of the experimental groups withdrawal of medication from the subjects used in the present study might have only enhanced further the contradictory results of the present study in connection with bilateral SCL differences.

General Discussion

The differentiation of schizophrenics into responder and nonresponder categories has both practical and theoretical significance. From a practical point of view if responders and nonresponders are generally characteristic of the population of hospitalized schizophrenics then some of the past research which has produced conflicting results may be explainable if a heterogenous, rather than a homogenous, model of schizophrenia is adopted.

From a theoretical standpoint the data of the responders and nonresponders in the present study is perhaps best seen as representing different forms of CNS dysfunction. The nonresponders may also be seen as having a more severe degree of CNS dysfunction as is evidenced by the neuropsychological data. In addition a number of other theoretical viewpoints are consistent with the data of the present study and that of other studies to an extent deserving of discussion. These include arousal theory, Broen's response interference and cue utilization theory, and the theory of protective or transmarginal inhibition.

Gruzelier and Venables (1972) view their nonresponder schizophrenics in terms of a progressive damage to the noradrenergic nerve endings of the fronto-temporal noradrenergic system (Stein & Wise, 1971). Interpreting the low SCLs and lower frequency of spontaneous fluctuations

as evidence of lower arousal in nonresponders, they cite evidence of stimulation of the amygdala in man producing increased behavioral and emotional activity and lesions producing the opposite effect (Chapman, Singh, Schroeder, & Fager, 1957; Narabayasli, Nagao, Salto, Yostrida, & Nagabata, 1963). Furthermore, they interpret the lack of electrodermal reactivity in nonresponders to neutral stimuli as comparable to a similar lack of electrodermal reactivity found in some amygdalectomized monkeys (Bagshaw & Benzies, 1968; Bagshaw, Kimble, & Pribram, 1965). Nonresponders who had no electrodermal reactivity to even signal stimuli were seen by Gruzelier and Venables as having a more severe dysfunction in the frontal regions of the noradrenergic frontolimbic system, much like the frontally brain-damaged patients described by Luria and Homskaya (1966, 1970). Citing evidence (Gruzelier & Venables, 1972) that hippocampal stimulation increases inhibition of electrodermal activity (Yokata & Fujimori, 1964; Yokata, Sato, & Fujimori, 1963) and the work by Kimble and his collaborators (Kimble, 1968) that lesions decrease inhibition, Gruzelier and Venables describe responders as having a temporal lobe dysfunction most likely involving the hippocampus. In a more recent paper (Gruzelier & Venables, 1975) these authors have discounted their previous view that responders and nonresponders have abnormally high and low arousal levels, respectively. However, their current (Gruzelier & Venables, 1973, 1974, 1975) interpretation of responders and nonresponders as indicative of hippocampal and amygdaloid damage, respectively, still has problems.

While it is true that amgdalectomy resulted in electrodermal nonresponding to habituation tones in monkeys, effects of the presentation of shock indicated that (1) the electrodermal threshold

sensitivity to shock was, if anything, decreased, and (2) the electrodermal reactivity appeared to occur in an all-or-none fashion in amygdalectomized monkeys whereas SCR amplitude in normal monkeys increased gradually with increasing shock. Gruzelier and Venables provide no evidence for the nature of SCR responding to shock in their patients. In the present study SCR amplitude of nonresponders during shock and (loud) startle stimuli, which produced startle reactions, was significantly attenuated, whereas SCR amplitude to neutral tones and spontaneous fluctuations, when they did occur, were comparable to those of the control group and responder group, thus providing no evidence for an all-or-none form of electrodermal reactivity or a decreased threshold of SCR reactivity.

Although lower SCLs, fewer spontaneous fluctuations and lower basal SCLs tend to support the early position of Gruzelier and Venables that nonresponders have lower arousal levels than normals, several results in the present study indicate, that in spite of the electrodermal data, nonresponders, as well as responders, are overaroused. First, the hypotensive effect of tranquilizers on blood pressure and compensatory increases in heart rate is a stable effect and differences in arousal level between the schizophrenic groups should be reflected in small (law of initial values effect) but observable differences in heart rate. However, heart rate was the same or higher for the nonresponder group compared with the responder group suggesting increased arousal for both groups. Secondly, respiration rate, another indicator of arousal but one not affected by tranquilizers (Tecce & Cole, 1972) is significantly higher for both schizophrenic groups compared with normals and further supports a view of high arousal for both schizophrenic groups. Thirdly, not all of the electrodermal data supports the early position of Gruzelier

and Venables. A concept of lower arousal for nonresponders would have difficulty in accounting for the SCR amplitude data of the present study. Low arousal in nonresponders should result in increased SCR amplitude during tasks producing large amplitude SCRs in normals with small (or no SCRs) during relatively stressless situations such as one involving repeated tones. In fact, the opposite results occurred. Finally, the changes in the duration or width of the "ascent windows" were comparable for normal controls and nonresponders: an increase during startle, breath inhalation, cognitive demands, shock delivery, and a decrease during habituation to tones and spontaneous fluctuations. However, what differed significantly was the lack of SCR reactivity or sympathetic inflow in the nonresponders during wider "ascent windows" but appropriate sympathetic inflow during narrower "ascent windows" occurring during habituation to tones and spontaneous fluctuations. A low arousal hypothesis for nonresponders would have difficulty in accounting for these results. However, the theories presented below would not.

Several theories of schizophrenia (for a review of theories see Magaro, 1974) can account for the results of the present study in respect to the data of both responders and nonresponders. Broen's (1968) theory based on schizophrenic behavioral research is one of the most encompassing available. Although Broen views the schizophrenic population as heterogenous in nature, he states that response interference is the most notable characteristic in the responding of most groups of unremitted schizophrenics. In general, when situations are responded to, the most prominent characteristics are (1) behavioral instability with (2) much of the variation being among the varied response tendencies that are also evoked in some strength in normal

subjects as well as in schizophrenics. To emphasize the lack of complete randomization among the alternate responses, Broen describes the response hierarchies of the schizophrenic as "partially collapsed". The term "partially" in this description is meant to indicate that while the organization of the response hierarchy is still there, the responses within it have more equal probabilities of occurrence than is the case for normals. In spite of the ability of the response interference principle to describe and account for the deficits found for many groups of schizophrenics, Broen notes again the heterogenous nature of the schizophrenic population: groups of schizophrenics overlap the normal distribution to a major extent, and some groups, including paranoid and possibly good premorbid chronic schizophrenics, seem to have less response interference than do other schizophrenics.

A second variable of primary importance in Broen's theory is the range of cue utilization. The heterogeneity of the schizophrenic population is even more obvious when considering the range of cue utilization in schizophrenics. Some acute and chronic paranoid schizophrenics seem to be responsive to a wider range of cues than that which normals respond to, while many chronic and poor premorbid (process) schizophrenics seem to respond to a reduced range of stimuli.[3] Broen (1968) interprets the wider than normal range of cue utilization in the acute schizophrenics as one aspect of the abnormal response interference.

Since Broen does not view acute or chronic schizophrenics as differing in either arousal or in response interference, an additional mechanism is postulated to account for the reduced range of cue utilization seen in chronic schizophrenics. He suggests two possible

mechanisms which might result in a reduced range of cue utilization. The first type of theory suggests that reduced range of cue utilization results from a learned and defensive style of attention in which reduced scanning may reduce the confusion which arises from extensive response interference that occurs in schizophrenics when there is wide scanning; this style of attention is used even at the cost of reduced information (Chapman, Chapman, & Miller, 1964; McGhie, 1970). A second type of theory suggests that the reduced cue utilization directly results from a basic disturbance in schizophrenia; for example, high arousal and consequent inhibitory feedback, an idea emphasized in the Pavlovian concept of transmarginal or protective inhibition (see Gray, 1964), or an abnormally slow ability to process information which results in much of the information being "lost" (Yates, 1966).

Broen's (1968) position on arousal is that both acute and chronic schizophrenics are highly aroused as supported by basal level psychophysiological response measures, whereas reactivity measures, especially electrodermal measures, do not support this position, particularly in the case of process or chronic schizophrenics (Reynolds, 1962; Williams, 1953). The evidence reviewed previously for responders and nonresponders with respect to arousal is certainly consistent with Broen's hypothesis: heart rate activity, and respiration rate did not differ between the responders and nonresponders and are consistent with an interpretation of high arousal for both groups of schizophrenics.

The basal measure of electrodermal levels in the present study are not, however, consistent with Broen's hypothesis. Although electrodermal measures are the least consistent measure of arousal compared with other psychophysiological measures, a simpler explanation may exist

for these results. The levels for both the nonresponders and responders were lower than those of the control group in the present study. However, the strong medication regime of the schizophrenic groups may be responsible for the lower levels of these groups since it is known that the major tranquilizers reduce electrodermal levels (Tecce & Cole, 1972). With no medication one or both schizophrenic groups may have had higher than normal electrodermal levels.

Excessive arousal alone could be used to account for the schizophrenic performance if one assumes an inverted U shaped relationship between arousal and performance (Yerkes & Dodson, 1908; Malmo, 1958). However, such an assumption would still not explain the dissociation of electrodermal changes from other psychophysiological response measures for the nonresponder group. Most theorists assume additional mechanisms to account for the disruption in performance with increases in arousal; these additional mechanisms could also handle the paradoxical electrodermal data. Hence, Meehl (1962) posits a greater equivalence of synaptic transmission probabilities, Stilson and Kopell (1964) suggest changed cortical synaptic transmission probabilities possibly resulting from abnormal cholinergic and/or adrenergic reactivity. Broen (1968) hypothesizes lower response ceiling strengths in schizophrenics compared with normals and a second hypothesis which would not require lower response ceiling strength. He cites Carlton's (1963) research concerning drug induced functional lesions of the septum in rats which suggests (1) that adrenaline may be one of the substances involved in mediating cortical arousal, (2) that both arousal related chemicals and acetycholine (ACH) are related to a collapse of response hierarchies, but (3) the effects are opposite, at least in structures where arousal

is initially at least moderate. In such situations, an increase in arousal related chemicals or a decrease in ACH tends to collapse response hierarchies in the sense that both tend to flatten the gradient of strengths of alternate responses. Food-deprived rats were trained to do a two-response sequence in order to obtain food: (a) push any one of 12 buttons on one wall, then (b) cross the chamber and press a lever. Under the normal experimental conditions, clear button-response hierarchies were developed. Amphetamines or scopolamine increased the alternate responses to other buttons (Carlton, 1963).

Broen's position, then is that both acute and chronic schizophrenic patients are characterized by a state of high arousal, a collapsing of response hierarchies, and a resultant deficit in their performance if tested in situations where successful performance to a stimulus depends on a successful inhibition of other responses in the subject's response hierarchies. Tests of range of cue utilization with multiple stimuli which demonstrate wider than normal cue utilization in acutes and paranoid chronics and a narrower than normal utilization of cues in chronic schizophrenics are dealt with in Broen's theory with an additional assumption; the chronic schizophrenic patient is assumed to have experienced disruption due to collapsed response hierarchies early in his disorder (an early poor premorbid state), and to have learned a defensive style of behavior and/or to have a brain disturbance which results in inhibitory feedback to protect him from the disruptive effects of high arousal.

On the basis of behavioral differences observed between acute and chronic schizophrenics and some indirect psychophysiological evidence reviewed by Broen (1968) it seems that responders in the present study

could be regarded as similar to that group of schizophrenics described by Broen (1968) as acute, chronic paranoid, highly aroused and responsive to a wider than normal range of cue utilization. The nonresponders in the present study resemble the chronic or poor premorbid (process) schizophrenics described by Broen (1968), who, although highly aroused, have a reduced range of cue utilization resulting in large selective attention deficits, possibly due to the presence of inhibitory feedback protecting the individual from the disruptive effects of constant excessive arousal.

The present study and work by Gruzelier and Venables demonstrates faster than normal electrodermal latency in responding schizophrenics during habituation tasks (Gruzelier & Venables, 1972, 1973) and during respiratory responses and startle reactions: all of these situations require no specific response on the part of the subject. However, as soon as these schizophrenic subjects are required to make even the simplest of behavioral discriminations, these subjects take longer to respond behaviorally and have longer than normal electrodermal response latencies. It seems possible that these schizophrenics, who are hypothesized to be more aroused than normals or to have an abnormality in cholinergic mechanisms, would demonstrate faster behavioral responses and shorter electrodermal latencies during simple stimulus situations where the association between the stimulus and the response is almost reflexive. Increases in stimulus complexity, on the other hand, hypothesized to result in increased response interference, should lead to slower behavioral performance and longer electrodermal latencies in these same schizophrenics, compared with normal controls. A higher number of spontaneous fluctuations in responders compared with normals

and nonresponders would be expected and would be consistent with the behavioral evidence of a broader range of cue utilization in acute and paranoid schizophrenics, compared with normal controls and chronic (nonresponder) schizophrenics (Broen, 1968).

Edelberg's (1970, 1972b) demonstration that SCR recovery measures are centrally mediated and are shorter during "goal-oriented" tasks led him to hypothesize that short recovery times, correlating with positive skin potential and absorption of sweat by skin, serve to prepare the skin for optimal tactile manipulability during the orienting response. With cognitive task demands or goal-oriented tasks in the present study both the normal control and the responding schizophrenics had short recovery times; however, during tasks of low cognitive demand, such as the repeated presentation of tones (task 2) the normals had very long recovery times, whereas the responding schizophrenics in the present study and in other studies (Ax & Bamford, 1971; Gruzelier & Venables, 1973) maintained short recovery times, a finding consistent with the hypotheses that these schizophrenics are highly aroused, have broader than normal ranges of cue utilization, and are unable to keep from responding to this habituation situation as if it were a goal oriented task necessitating tactile preparation for the execution of a response.

Variations in the width of the SCR "ascent windows" with task demands has not been investigated by other researchers. The results in the present study suggest that the SCR changes to variations in task demands appear to result from two mechanisms. First, the width of the "ascent window" for SCRs appears to change according to the arousing properties of the stimulus. Sudden noise or cognitive demands

occurring during a state of low electrodermal activity results in very wide "ascent windows" which normally also contain an SCR with a very steep ascent slope. Secondly, the actual slope of SCRs also appears to vary with the demands of the task, irrespective of their amplitude. The smaller, multiple SCRs, occurring after the initial large amplitude SCR to cognitive tasks have steep ascent slopes whereas the smaller multiple SCRs occurring during a state of rest (i.e., spontaneous fluctuations or SCRs to tones during a habituation task) have very gentle ascent slopes. Therefore the significantly narrower SCR "ascent windows" observed in responding schizophrenics in the present study to respiration changes and startle responses would be consistent with an interpretation that, compared with normals, the responding schizophrenics are more aroused and are responding to the task as though it had goal orienting properties.

Broen's suggestion that chronic schizophrenics have a learned and defensive style of attention and/or an inhibitory feedback system, with deficits in the range of cue utilization can account for the findings of electrodermal activity in nonresponders in the present study. In a study of over-inclusion of stimulus materials, chronics, who showed less over-inclusion than did acutes, compared with normals, were the most retarded or slow in selecting their choices (Payne, 1966). The longer SCR latency of nonresponding schizophrenics (chronics) in the present study compared with controls for tasks requiring behavioral responses as well as for those tasks which did not, is consistent with the hypothesis that chronic (nonresponder) schizophrenics, as a result of their limited and reduced scanning ability, have an abnormally slow ability to process information. The fewer spontaneous fluctuations

and SCRs to repeated tones reported to occur in nonresponders is also consistent with the evidence of limited processing of information by the chronic or process group of schizophrenics. The evidence of attenuated SCR reactivity to stressful tasks (rather than the expected increases) found in the present study is also consistent with the hypothesis of a protective inhibitory feedback system which protects the subject from the effects of stress. Furthermore, the lack of differences between the nonresponders and normals in variations of width of SCR "ascent windows" suggests that the variations in cognitive demands of the tasks were processed by the nonresponders as is evidenced by the modulation of the ascent windows widths. What differed between the nonresponders and the normals was some sort of protective mechanism, perhaps, which allowed normal sized SCRs to stressless situations, but limited the amplitude of the SCR during stressful situations.

The paradoxical decrease of electrodermal reactivity in nonresponding schizophrenics is not a unique finding to this study. Several studies reviewed by Broen (1968) on physiological measures of arousal demonstrated that process or chronic schizophrenics showed less change in electrodermal reactivity (and other physiological measures) to increases in stress compared with other schizophrenics and with normals (Reynolds, 1962; Williams, 1953). Silverman (1972) demonstrated similar effects in some schizophrenics with the averaged visual evoked potential response. Smith (1967) reported smaller electrodermal responses to 110 db tones than to 70 db tones in withdrawn schizophrenics, whereas normals and more active schizophrenics had the expected increase in electrodermal responsivity to increases in stimulus intensity. Depue and Fowles (1976), Venables (1971), and Gray (1964) have all invoked the Pavlovian concept

of protective inhibition to account for these results.

Broen's position on internal or protective inhibition differs somewhat from that of Gray or Depue and Fowles. In Broen's theory (1968) the disturbance associated with excessive arousal is experienced early in a poor premorbid schizophrenic resulting in an early formation of protective inhibition and defensive behavioral style of uniformly limiting stimulus input; the acute schizophrenic has no such protective inhibition. Broen cites Fenz's work with parachutists (Fenz & Epstein, 1967) and Lacey's work with heart rate changes during receptive or anticipatory sets and defensive sets (Lacey, 1959) as evidence of how physiological systems can be controlled through experience in normals. Thus, Broen postulates that the poor premorbid schizophrenic learns, through repeated experience, to trigger the protective inhibition earlier and earlier in a chain of stimulus events leading to stress, much as the experienced parachutist does in the sequence of events leading to a jump.

Although the Russian theory and research (see Gray, 1964) emphasizes the predominance of low cortical activation in schizophrenics which is comparable to Broen's position at this point, the interpretation of the effects of protective inhibition are not assumed to be uniform as is the case in Broen's position. Low levels of protective inhibition are seen to decrease, rather than increase, the suppression of normally inhibited responses in acute and paranoid schizophrenics. With further increases in levels of protective inhibition (producing the low cortical activation and increases in attentional deficits) the Russian position and Broen's position are similar in their prediction of behavioral effects.

A practical implication of this position is that psychophysiological response measures may provide the necessary independent measure for categorizing heterogenous schizophrenics into the appropriate categories for behavioral research on schizophrenics. The finding that nonresponders in the present study were significantly more often described as chronic did not, however, mitigate a large overlapping with the hospitalization duration of the responder group. One example of better differentiation of the schizophrenics in the present study is the case of a very chronic paranoid who was categorized as a responder much like the young acute reactive schizophrenic which is consistent with Broen's hypotheses. Another example is the better differentiation of schizophrenic's performance on the Halstead-Reitan neuropsychological test battery when using psychophysiological variables for sorting rather than chronicity of illness criteria.

The electrodermal and neuropsychological results of the present study suggest two very different types of CNS dysfunction for the nonresponding and responding schizophrenics. Currently, the present author favors the suggestions of Gruzelier and Venables (1972, 1973) as to what types of dysfunction exist in these two schizophrenic populations. Their position is similar to that presented by Kornetsky and Mirsky (1966), with respect to the areas of the brain thought to be involved. Kornetsky and Mirsky suggest that in the chronic nuclear cases, one is examining a schizophrenia of long standing that may be associated with diffuse frontal lobe damage including destruction of tissue in ventral and orbital frontal areas and some associated subcortical structures such as the medialis dorsalis of the thalamus and the head of the caudate nucleus. They describe this group behaviorally

as stuporous, blunted, and withdrawn. In the episodic, paranoid group of schizophrenics the damage may be preferentially found in the septal, hippocampal, and temporal lobe areas, although some frontal damage is also thought to occur. This latter population of schizophrenics is more likely to be characterized as having personality difficulties and described as aggressive, assaultive, fragmented, and bizarre (Mirsky, 1969).

The results of tests in the present study that are most sensitive to frontal lobe dysfunction in the Halstead-Reitan battery are certainly supportive of Kornetsky and Mirsky's position. The mean performance of the controls, responders, and nonresponders on the Halstead Category test was 27.5, 55.0, and 115.2, respectively. The effects of hippocampal lesions on electrodermal reactivity in monkeys and the similarity to responding in certain schizophrenics (Bagshaw & Kimble, 1972) is also consistent with Kornetsky and Mirsky's views as well as the suggestion by Broen (1968) that collapsing of response hierarchies is possible with deficits of ACH from septal disturbances.

A difficulty remains with the latter speculation of equating responding schizophrenics as evidencing hippocampal and/or septal dysfunction. In animal research on the behavioral effects of lesions in these areas, the deficit is best described as a perservation of responding (Douglas, 1967) or an inability to shift attention, resulting in inordinate indistractibility (Hendrickson, Kimble, & Kimble, 1969). Schizophrenics, on the other hand, are seen as inordinately distractible, or, as Meehl puts it "...Defective inhibition is the most direct and uncomplicated neurologizing of the schizophrenic cognitive slippage. Schizoid cognitive slippage is neither an incapacity to link, nor is it

an unhealthy over capacity to link; rather, it seems to be a defective control over associations which are also accessible to the healthy (or in dreams, wit, psychoanalytic free association, and certain forms of creative work) but which are normally 'edited out' or automatically suppressed by those superordinate monitoring assembly systems we lump together under the term set" (1962, p.834).

Whether the behavioral syndrome postulated in the animal research on hippocampal deficits is actually different from that postulated for schizophrenics or is a result of the tasks used, or in the complexity of the organisms tested, remains to be seen. Certainly much of the behavioral deficits in schizophrenics reviewed by Broen (1968) such as Fey's (1951) use of the Wisconsin Card Sorting Task, Lang and Luoto's (1962) work with training in competing responses, or Bzhalava's (1965) research on fixation of set, are consistent with a concept of disinhibition of prepotent responses.

FOOTNOTES

[1]Schneider's symptoms of the first rank: audible thoughts, voices heard commenting on one's actions; the experience of influences playing on the body (somatic passivity experiences); thought-withdrawal and other interferences with thought; diffusion of thought; delusional perception and all feelings, impulses (drives) and volitional acts that are experienced by the patient as the work or influence of others.

[2]For a diagnosis of schizophrenia A through C are required. A. Both of the following are necessary: (1) a chronic illness with at least six months of symptoms prior to the index evaluation without return to the premorbid level of psychosocial adjustment, and (2) absence of a period of depressive or manic symptoms sufficient to qualify for affective disorder or probable affective disorder. B. The patient must have at least one of the following: (1) delusions or hallucinations without significant perplexity or disorientation associated with them and (2) verbal production that makes communication difficult because of a lack of logical or understandable organization. C. At least three of the following manifestations must be present for a diagnosis of "definite" schizophrenia and two for a diagnosis of "probable" schizophrenia: (1) single, (2) poor premorbid social adjustment or work history, (3) family history of schizophrenia, (4) absence of alcoholism or drug abuse within one year of the onset of psychosis, and (5) onset of illness prior to age 40.

[3]Broen does not view the heterogeneity of schizophrenic groups as fitting into a continuum from acute to chronic illness with certain exceptions such as chronic paranoid schizophrenics. Rather, he views

the heterogenous groups as discrete groups who may represent different forms or types of defects. Thus, the chronic as well as the poor premorbid (process) schizophrenic who is acute by definition of hospitalization length, shows a different type of defect than does the acute (reactive) schizophrenic, who seldom becomes chronic, or the chronic paranoid schizophrenic.

BIBLIOGRAPHY

Alexander, A.A. Psychophysiological concepts of psychopathology. In N.S. Greenfield & R.A. Sternbach (Eds.), Handbook of psychophysiology. New York: Holt, Reinhart and Winston, Inc., 1972, 925-964.

Anonkhin, P.K. The problem of the center and periphery in modern physiology of the higher nervous activity. Problems of higher nervous activity. Gorki, 1935, 9-70. (In Russian)

Anonkhin, P.K. The features of the afferent apparatus of the conditional reflex. Vopr. Psikholog., 1955, 6. (In Russian)

Ax, A.F., & Bamford, J.L. The GSR recovery limb in chronic schizophrenia. Psychophysiology, 1971, 7, 145-147.

Bagshaw, M.H., & Benzies, S. Multiple measures of the orienting reaction and their dissociation after amygdalectomy in monkeys. Experimental Neurology, 1968, 20, 175-187..

Bagshaw, M.H., & Kimble, D.P. Bimodal EDR orienting response characteristics of limbic lesioned monkeys: Correlates with schizophrenic patients. (Paper read before American Society for Psychophysiological Research, 1972.)

Bagshaw, M.H., Kimble, D.P., & Pribram, K.H. The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus, and inferotemporal cortex. Neuropsychologia, 1965, 3, 111-119.

Benton, A.L. Contributions to clinical neuropsychology. Chicago: Adline, 1969.

Bernstein, A.S. The phasic electrodermal orienting response in chronic schizophrenics. II Response to auditory signals of varying intensity. Journal of Abnormal Psychology, 1970, 75, 146-156.

Bernstein, N.A. Problems of the relation of coordination and localization. Archives of Biological Sciences, 1935, 38, 7. (In Russian)

Bernstein, N.A. On the structure of movements. Moscow: Medical Publishing House, 1947. (In Russian)

Bernstein, N.A. Actual problems on the physiology of activity. Problems of Cybernetics, 1961, VI, Moscow. (In Russian)

Blum, R.H. Alpha-rhythm responsiveness in normal, schizophrenic, and brain damaged persons. Science, 1957, 126, 749-750.

Bradley, C. Behavior disturbance in epileptic children. Journal of the American Medical Association, 1951, 146, 436-441.

Broen, W.E. Jr. Schizophrenia: Research and theory. New York: Academic Press, 1968.

Bull, R.H.C., & Gale, M.A. Electrodermal activity recorded concomitantly from the subject's two hands. Psychophysiology, 1975, 12(1), 94-97.

Bzhalava, I.T. On the psychopathology of fixated set in epilepsy and schizophrenia. Cortex, 1965, 1(4), 493.

Cannon, W.B. The wisdom of the body. New York: Norton, 1932.

Carlton, P.L. Cholinergic mechanisms in the control of behavior by the brain. Psychological Review, 1963, 70, 19-39.

Chapman, L.J., Chapman, J.P., & Miller, G.A. A theory of verbal behavior in schizophrenia. In B.A. Maher (Ed.), Progress in experimental personality research. New York: Academic Press, 1964, 1, 49-77.

Chapman, W.P., Singh, M.M., Schroeder, H.R., & Fager, C. Temporal lobe epilepsy: Brief review. Responses on electrical stimulation of the amygdaloid region in six patients. American Journal of Medicine,

1957, 23, 107-119.

Davidoff, R.A., & McDonald, D.G. Alpha blocking and autonomic responses in neurological patients. Archives of Neurology, 1964, 10, 283-292.

Depue, R.A., & Fowles, D.C. Electrodermal activity as an index of arousal in schizophrenics. Psychological Bulletin, 1973, 79, 233-238.

Depue, R.A., & Fowles, D.C. Electrodermal activity and schizophrenia: The problem of stimulus intensity modulation. Psychological Bulletin, 1976, 83, 192-193.

Diamond, S. The double brain. Edinburgh: Churchill Livingstone, 1972.

Douglas, R. Hippocampus and behavior. Psychological Bulletin, 67, 416-442.

Dykman, R.A., Reese, W.G., Galbrecht, C.R., Ackerman, P.T., & Sunderman, R.S. Autonomic responses in psychiatric patients. Annals of the New York Academy of Sciences, 1968, 147, 237-303.

Edelberg, R. The information content of the recovery limb of the electrodermal response. Psychophysiology, 1970, 6, 527-539.

Edelberg, R. Electrical activity of the skin: Its measurement and uses in psychophysiology. In N.S. Greenfield & R.A. Sternbach (Eds.), Handbook of psychophysiology. New York: Holt, Rinehart, and Winston, Inc., 1972a, 367-409.

Edelberg, R. Electrodermal recovery rate, goal-orientation, and aversion. Psychophysiology, 1972b, 9, 512-520.

Feighner, J.P., Robins, E., Guze, S.B., Woodruff, R.A., Winokur, G., & Munoz, R. Diagnostic criteria for use in psychiatric research. Archives of General Psychiatry, 1972, 26, 57-63.

Fenz, W.D., & Epstein, S. Gradients of physiological arousal in parachutists as a function of an approaching jump. Psychosomatic Medicine, 1967, 29, 35-51.

Fey, E.T. The performance of young schizophrenics and young normals on the Wisconsin Card Sorting Test. Journal of Consulting Psychology, 1951, 15, 311-319.

Filskov, S.B., & Goldstein, S.G. Diagnostic validity of the Halstead-Reitan Neuropsychological Battery. Journal of Consulting and Clinical Psychology, 1974, 42, 382-388.

Flor-Henry, P. Psychosis and temporal lobe epilepsy. A controlled investigation. Epilepsia, 1969, 10, 363-395.

Flor-Henry, P. Cerebral dominance: Functional disturbance in schizophrenic, the affective psychoses, neuroses, and psychopathy: A heuristic hypothesis. (Paper presented at the joint meeting of the Canadian Psychiatric Association, Quebec Psychiatric Association, Royal College of Psychiatrists, Montreal, Canada, June 1972.)

Flor-Henry, P. Hemispheric dysfunction and psychopathology. (Paper prepared for McGill University Symposium of "Transformations of Consciousness," Montreal, October 1973.)

Flor-Henry, P. Psychosis, neurosis and epilepsy. Developmental and gender-related effects and their aetiological contribution. British Journal of Psychiatry, 1974, 124, 144-150.

Flor-Henry, P., Koles, Z.J., Bo-Lassen, P., & Yeudall, L.T. Studies of the functional psychoses: Power spectral analysis. International Research Communications System: Medical Science, 1975, 3, 87.

Flor-Henry, P., Yeudall, L.T., Stefanyk, W., & Howarth, B. The neuropsychological correlates of the functional psychoses.

International Research Communications System: Medical Science, 1975, 3, 34.

Galin, O., & Ornstein, R. Lateral specialization of cognitive mode: An EEG study. Psychophysiology, 1972, 9, 412-418.

Gazzaniga, M.S. The bisected brain. New York: Appleton-Century-Crofts, 1970.

Gazzaniga, M.S., Bogen, J.E., & Sperry, R.W. Observations on visual perception after disconnection of the cerebral hemispheres in man. Brain, 1965, 8, 221-236.

Gray, J.A. Pavlov's typology. Recent theoretical and experimental developments from the laboratory of B.M. Teplov. New York: Macmillan, 1964.

Gruzelier, J.H. Bilateral asymmetry of skin conductance orienting activity and levels in schizophrenics. Journal of Biological Psychology, 1973, 1, 21-41.

Gruzelier, J.H., & Venables, P.H. Two flash thresholds, heart rate, skin temperature, and blood pressure in schizophrenics with and without skin conductance orienting responses. (Paper presented at the meeting of the British Psychological Society, London, December 1971.)

Gruzelier, J.H., & Venables, P.H. Skin conductance orienting activity in a heterogeneous sample of schizophrenics: Possible evidence of limbic dysfunction. Journal of Nervous and Mental Disease, 1972, 155(4), 277-287.

Gruzelier, J.H., & Venables, P.H. Skin conductance responses to tones with and without attentional significance in schizophrenics and nonschizophrenic psychiatric patients. Neuropsychologia, 1973, 11,

221-230.

Gruzelier, J.H., & Venables, P.H. Bimodality and lateral asymmetry of skin conductance orienting activity in schizophrenics: Replication and evidence of lateral asymmetry in patients with depression and disorders of personality. Biological Psychiatry, 1974, 8, 55-73.

Gruzelier, J.H., & Venables, P.H. Evidence of high and low arousal in schizophrenics. Psychophysiology, 1975, 12, 66-73.

Gunderson, E.K. Autonomic balance in schizophrenia. Unpublished doctoral dissertation, University of California, Los Angeles, 1953. (Cited in Tecce & Cole, 1972.)

Guttman, L., & List, C.F. Die nervosen leitungsbahnen der schweissekretion beim menschen. Deutsche Zeitschrift fur Nervenheilkunde, 1928, 167, 61-71.

Hendrickson, C.W., Kimble, R.J., & Kimble, D.P. Hippocampal lesions and the orienting response. Journal of Comparative and Physiological Psychology, 1969, 67, 220-227.

Hoff, E.C., Kell, J.R. Jr., & Carroll, M.N. Jr. Effects of cortical stimulation and lesions on cardiovascular function. Physiological Reviews, 1963, 43, 68-114.

Holloway, F.A., & Parsons, O.A. Unilateral brain damage and bilateral skin conductance levels in humans. Psychophysiology, 1969, 6(2), 138-148.

Holloway, F.A., & Parsons, O.A. Habituation of the orienting reflex in brain damaged patients. Psychophysiology, 1971, 8(5), 623-634.

Ingram, W.R. Central autonomic mechanism. In J. Field, H.W. Magoun & V.E. Hall (Eds.), Handbook of physiology, section 1, neurophysiology, Vol. 2. Washington D.C., American Physiological

Society, 1960, 951-978.

Jordan, L.S. Electrodermal activity in schizophrenics: Further considerations. Psychological Bulletin, 1974, 81, 85-91.

Jurko, M., Jost, H., & Hill, T.S. Pathology of the energy system: An experimental study of physiological adaptive capacities in a nonpatient, a psycho-neurotic, and an early paranoid schizophrenic group. Journal of Psychology, 1952, 33, 183-198.

Kimble, D.P. Hippocampus and internal inhibition. Psychological Bulletin, 1968, 70, 285-295.

Kornetsky, C., & Mirsky, A.F. On certain psychopharmacological and physiological differences between schizophrenic and normal persons. Psychopharmacologia, 1966, 8, 309-318.

Lacey, J.I. Psychophysiological approaches to the evaluation of psychotherapeutic process and outcome. In E.A. Rubinstein and M.B. Parloff (Eds.), Research in psychotherapy. Washington, D.C.: National Publishing Company, 1959, 160-208.

Lacey, J.I., Bateman, D.E., & VanLehn, R. An experimental study. Psychosomatic Medicine, 1953, 15, 8-21.

Lacey, J.I., & VanLehn, R. Differential emphasis in somatic response to stress. Psychosomatic Medicine, 1952, 14, 73-81.

Lang, P.J., & Hnatiow, J. Stimulus repetition and the heart rate response. Journal of Comparative and Physiological Psychology, 1962, 55, 781-785.

Lang, P.J., & Luoto, K. Mediation and associative facilitation in neurotic, psychotic, and normal subjects. Journal of Abnormal and Social Psychology, 1962, 64, 113-120.

Lovallo, W. The cold pressor test and autonomic function: A review

and integration. Psychophysiology, 1975, 12, 268-282.

Luria, A.R. Human brain and psychological processes. Harper & Row, New York & London, 1966.

Luria, A.R., & Homskaya, E. Disturbance of the regulating activity lesion of the frontal lobes of the brain. In The human brain and psychic process. Izd APN RSFSR: Moscow, 1963, 430-453.

Luria, A.R., & Homskaya, E. Disturbance of action control in frontal lobe lesions. In A.R. Luria (Ed.), Human brain and psychological processes. Harper & Row, New York & London, 1966.

Luria, A.R., & Homskaya, E. Frontal lobe and the regulation of arousal processes. In D. Mostofsky (Ed.), Attention: Contemporary theory and research. New York: Appleton Century Croft, 1970.

Lykkens, D.T., & Venables, P.H. Direct measurement of skin conductance: A proposal for standardization. Psychophysiology, 1971, 8, 656-672.

Magaro, P.A. Theories of the schizophrenic performance deficit: An integration theory synthesis. In B.A. Maher (Ed.), Progress in experimental personality research. New York: Academic Press, 1974, 7, 149-208.

Malmo, R.B. Measurement of drive: An unsolved problem in psychology. In M.R. Jones (Ed.), Nebraska Symposium on Motivation. Lincoln: University of Nebraska Press, 1958.

McDonald, R.L., & Gynther, M.D. Effects of verbal stimuli on autonomic responsivity of medicated and non-medicated schizophrenics and character disorders. Journal of Genetic Psychology, 1962, 66, 287-299.

McGhie, A. Attention and perception in schizophrenia. In B.A. Maher (Ed.), Progress in experimental personality research. New York:

Academic Press, 1970, 5, 1-38.

McNemar, Q. Psychological statistics. New York: John Wiley & Sons, 1957, 202.

Mednick, S.A. Breakdown in individuals at high risk for schizophrenia: Possible predispositional perinatal factors. Mental Hygiene, 1970, 54, 50-63.

Mednick, S.A., & Schulsinger, F. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. In D. Rosenthal and S.S. Kety (Eds.), The transmission of schizophrenia. London: Pergamon Press, 1968.

Meehl, P.E. Schizotaxia, schizotypy, schizophrenia. American Psychologist, 1962, 17, 827-838.

Milner, B., & Taylor, L. Right-hemisphere superiority in tactile pattern-recognition after cerebral commissurotomy: Evidence for non-verbal memory. Neuropsychologia, 1972, 10, 1-15.

Mirsky, A.F. Neuropsychological bases of schizophrenia. Annual Review of Psychology, 1969, 20, 321-348.

Mountcastle, V.B. (Ed.) Interhemispheric relations and cerebral dominance. Baltimore: John Hopkins Press, 1962.

Narabayasli, H., Nagao, T., Salto, Y., Yostrida, M., & Nagabata, M. Stereotaxic amygdalotomy for behavior disorders. Archives of Neurology, 1963, 9, 1-16.

Obrist, P.A., Webb, R.A., & Sutterer, J.R. Heart rate and somatic changes during aversive conditioning and a simple reaction time task. Psychophysiology, 1969, 5, 696-723.

Parsons, O.A. Clinical neuropsychology. In C.D. Spielberger (Ed.), Current topics in clinical and community psychology. New York:

Academic Press, 1970, 2, 1-60.

Parsons, O.A., & Chandler, P. Electrodermal indicants of arousal in brain damage: Cross-validated findings. Psychophysiology, 1969, 5, 644-659.

Payne, R.W. The measurement and significance of overinclusive thinking and retardation in schizophrenic patients. In P.H. Hoch & J. Zubin (Eds.), Psychopathology of schizophrenia. New York: Grune & Stratton, 1966, 77-97.

Penfield, W. The neurophysiological basis of thought. In J.G. Howells (Ed.), Modern perspectives in world psychiatry. New York: Brunner/Mazel, 1971, 314-349.

Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and performance. Journal of Experimental Psychology, 1961, 62, 523-531.

Reitan, R.M., & Davison, L. (Eds.), Clinical neuropsychology: Current status and applications. New York: John Wiley & Sons, 1974.

Reynolds, D.J. An investigation of the somatic response system in chronic schizophrenia. Unpublished doctoral dissertation, University of Pittsburgh, 1962 (Cited in Broen, 1968).

Rickles, W.H. Jr. Central nervous system substrates of some psychophysiological variables. In N.S. Greenfield & R.A. Sternbach (Eds.), Handbook of psychophysiology. New York: Holt, Reinhart & Winston, Inc., 1972, 93-117.

Schneider, K. Klinische Psychopathologie. English translation, Clinical psychopathology (5th edition). New York: Grune & Stratton, 1959.

Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. Journal of Experimental

Psychology, 1959, 58, 117-128.

Silverman, J. Stimulus intensity modulation and psychological disease. Psychopharmacologia, 1972, 24, 42-80.

Slater, E., Beard, A.W., & Glithero, E. The schizophrenic-like psychoses of epilepsy. The British Journal of Psychiatry, 1963, 109, 95-150.

Smith, B.D. Habituation and spontaneous recovery of skin conductance and heart rate in schizophrenics and controls as a function of repeated tone presentations (Doctoral dissertation, University of Massachusetts, 1967). Dissertation Abstracts International, 1967, 28, 3068B, University Microfilms No. 67 17.

Sokolov, E.N. Perception and the conditioned reflex. New York: Pergamon Press, 1963.

Sourek, K. The nervous control of skin potentials in man. Pravda: Nakladatelstvi Cheskoslovenske Academie Ved, 1965.

Stein, L., & Wise, C.D. Possible etiology of schizophrenia. Progressive damage to the noradrenergic reward system by 6-hydroxydopamine. Science, 1971, 171, 1032-1036.

Stern, J.A., Surphlis, W., & Koff, E. Electrodermal responsiveness as related to psychiatric diagnosis and prognosis. Psychophysiology, 1965, 2, 51-61.

Stilson, D.W., & Kopell, B.S. The recognition of visual signals in the presence of visual noise by psychiatric patients. Journal of Nervous and Mental Disease, 1964, 139, 209-221.

Tecce, J.J., & Cole, J.O. Psychophysiologic responses of schizophrenics to drugs. Psychopharmacologia (Berl.), 1972, 24, 159-200.

Turskey, B. Presidential address. Physical, physiological and

psychological factors that affect pain reaction to electric shock. Psychophysiology, 1973, 11, 95-112.

Venables, P.H. Perception, attention, and arousal in schizophrenia. Paper presented at the meeting of the Finnish Psychiatric Association, Tempene March 18-20, 1971. (Cited in Depue & Fowles, 1976.)

Venables, P.H. Input regulation and psychopathology. In M. Hammer, K. Salzinger, & S. Sutton (Eds.), Towards a science of psychopathology. New York: Wiley, 1972.

Venables, P.H. A psychophysiological approach to research in schizophrenia. In D.C. Fowles (Ed.), Clinical applications of psychophysiology. New York: Columbia University Press, 1975, 106-138.

Wells, C.E. Response of alpha waves to light in neurologic disease. Archives of Neurology, 1962, 6, 478-491.

White, L. Organic factors and psychophysiology in childhood schizophrenia. Psychological Bulletin, 1974, 81, 238-255.

Wilder, J. Modern psychophysiology and the law of initial value. American Journal of Psychotherapy, 1958, 12, 199-221.

Williams, M. Psychophysiological responsiveness to psychological stress in early chronic schizophrenic reactions. Psychosomatic Medicine, 1953, 15, 456-462.

Wing, J.K., Birley, J.L.T., Cooper, J.E., Graham, P., & Isaacs, A.D. Reliability of a procedure for measuring and classifying "present psychiatric state." British Journal of Psychiatry, 1967, 113, 499-515.

Yates, A.J. Psychological deficit. In P.R. Farnsworth (Ed.), Annual review of psychology. Palo Alto: Annual Reviews Inc., 1966, 111-144.

Yerkes, R.M., & Dodson, J.D. The relation of strength of stimulus to rapidity of habit formation. Journal of Comparative Neurology, 1908, 18, 459-482.

Yeudall, L.T. Neuropsychological analysis of alcoholics with a psychiatric disorder. Accepted by Journal of Studies on Alcohol, 1976.

Yeudall, L.T., & Flor-Henry, P. Lateralized cerebral dysfunction in depression and criminal psychopath: A neuropsychological analysis. Submitted to the British Journal of Psychiatry, 1975.

Yokata, T., & Fujimori, B. Effects of brain stem stimulation upon hippocampal electrical activity, somatomotor reflexes and autonomic functions. Electroencephalography and Clinical Neurophysiology, 1964, 16, 375-382.

Yokata, T., Sato, A., & Fujimori, B. Inhibition of sympathetic activity by stimulation of limbic systems. Japanese Journal of Physiology, 1963, 18, 138-154.

Zangwill, O.L. The cerebral localization of cerebral function. Advances in Science, 1963, 20, 1-10.

APPENDIX A

Summary of Analyses of Electrodermal Amplitudes

Appendix A

TABLE A

Means for Normals (NC), Responders (R) and Nonresponders (NR) on SCR amplitudes in fig. 3 and Scheffe test of significance ($p < .05$). Any two means not underscored by the same line are significantly different. Any two means underscored by the same line are not significantly different.

	HANDS					
	RIGHT			LEFT		
1.	R	NC	NR	R	NC	NR
	.65	.60	.10	.70	.57	.06
2.	NC	R	NR	NC	R	NR
	.57	.38	.03	.56	.44	.02
3.	NC	R	NR	NC	R	NR
	.51	.48	.10	.52	.40	.10
4.	R	NC	NR	R	NC	NR
	.19	.10	.05	.12	.08	.02
5.	NC	R	NR	NC	R	NR
	.15	.08	.005	.15	.09	.006
6.	NC	R	NR	NC	R	NR
	.18	.10	.003	.18	.13	.005
7.	NC	R	NR	NC	R	NR
	.29	.21	.02	.29	.21	.02
8.	NC	R	NR	R	NC	NR
	.05	.03	.001	.04	.04	.003
9.	NC	R	NR	NC	R	NR
	.05	.05	.008	.07	.06	.006
10.	NC	R	NR	NC	R	NR
	.09	.05	.004	.09	.06	.005
11.	NC	R	NR	NC	R	NR
	.26	.19	.08	.26	.19	.06

TABLE B

Mean amplitude correlated t probabilities between eleven situations in Figure 3 for normal controls.

	Hand	2	3	4	5	6	7	8	9	10	11
1.	Right	.77	.284	.000	.000	.000	.000	.000	.000	.000	.000
	Left	.919	.498	.000	.000	.000	.001	.000	.000	.000	.000
2.			.413	.000	.000	.000	.002	.000	.000	.000	.000
			.519	.000	.000	.000	.004	.000	.000	.000	.000
3.				.000	.000	.001	.006	.000	.000	.000	.002
				.000	.000	.000	.005	.000	.000	.000	.000
4.					.093	.009	.000	.014	.028	.957	.000
					.047	.012	.000	.022	.706	.584	.000
5.						.041	.001	.006	.007	.171	.001
						.111	.001	.007	.026	.133	.001
6.							.025	.001	.001	.032	.039
							.014	.002	.009	.040	.030
7.								.000	.000	.000	.346
								.000	.000	.000	.229
8.									.466	.002	.000
									.003	.002	.000
9.										.000	.000
										.076	.000
10.											.000
											.000

TABLE C

Mean amplitude correlated t probabilities between eleven situations in Figure 3 for responding schizophrenics.

	Hand	2	3	4	5	6	7	8	9	10	11
1.	Right	.016	.042	.047	.008	.008	.030	.005	.007	.005	.024
	Left	.026	.225	.014	.012	.010	.039	.008	.010	.008	.023
2.			.607	.240	.024	.031	.211	.016	.029	.021	.173
			.818	.054	.036	.054	.173	.023	.030	.024	.115
3.				.196	.038	.051	.041	.022	.016	.030	.034
				.076	.040	.105	.055	.021	.018	.027	.047
4.					.234	.269	.860	.083	.159	.143	.990
					.262	.801	.193	.010	.076	.062	.317
5.						.326	.056	.025	.349	.197	.095
						.295	.035	.090	.336	.126	.081
6.							.122	.005	.182	.013	.147
							.265	.109	.203	.137	.393
7.								.011	.003	.031	.632
								.009	.008	.016	.344
8.									.448	.099	.016
									.338	.419	.022
9.										.871	.004
										.933	.015
10.											.036
											.028

TABLE D

Mean amplitude correlated t probabilities between eleven situations in Figure 3 for nonresponding schizophrenics.

	Hand	2	3	4	5	6	7	8	9	10	11
1.	Right	.150	.978	.346	.071	.064	.094	.060	.058	.069	.526
	Left	.232	.227	.212	.080	.072	.163	.060	.037	.074	.922
2.			.157	.504	.109	.087	.826	.066	.228	.051	.310
			.072	1.000	.211	.222	.857	.190	.393	.164	.163
3.				.374	.054	.049	.081	.048	.041	.053	.415
				.092	.033	.031	.046	.029	.025	.031	.185
4.					.189	.161	.482	.151	.227	.181	.602
					.298	.233	.948	.160	.319	.270	.264
5.						.170	.044	.197	.668	.685	.148
						.351	.104	.197	1.000	.351	.070
6.							.031	.351	.351	.685	.136
							.116	.170	.844	1.000	.067
7.								.023	.048	.022	.215
								.100	.210	.075	.095
8.									.250	.351	.130
									.504	.351	.060
9.										.549	.121
										.857	.045
10.											.142
											.063

Analysis of SCR Amplitude during Habituation/Dishabituation (Task 2)

The mean amplitude for each trial including zero amplitude are illustrated in Figure 1 for all three groups. Tests of groups by trials effects of SCR amplitude were carried out on data for both hands with two-factor analyses of variance, with 18 repeated measures (Trials 1-18) as one factor. The differences in mean SCR amplitude between groups approached significance for the right hand (F (2,29) = 2.6, $p < .09$) and reached significance for the left hand (F (2,29 = 3.91, $p < .05$). The repeated measures trials effect was significant for both hands, right hand F (17,493) = 6.55, $p < .001$; left hand F (17,493) = 6.08, $p < .001$. The trials by groups interactions was also significant for both hands, right hand F (34,293) = 2.04, $p < .001$; left hand F (34,493) = 2.04, $p < .001$.

Scheffe multiple comparisons (Table E) and correlated t tests between trials for each group (Tables, F, G, H) were performed. The significant groups by repeated measures interaction for Task 2 was interpreted as resulting from lower overall SCR amplitudes as well as less variation in amplitude across trials for the nonresponders compared with the controls and responding schizophrenics. However, as the Scheffe comparisons of Task 2 demonstrate (Table E) the three groups do not differ significantly in SCR amplitude for most tone trials which is in conflict with the results reported by Gruzelier and Venables (1972).

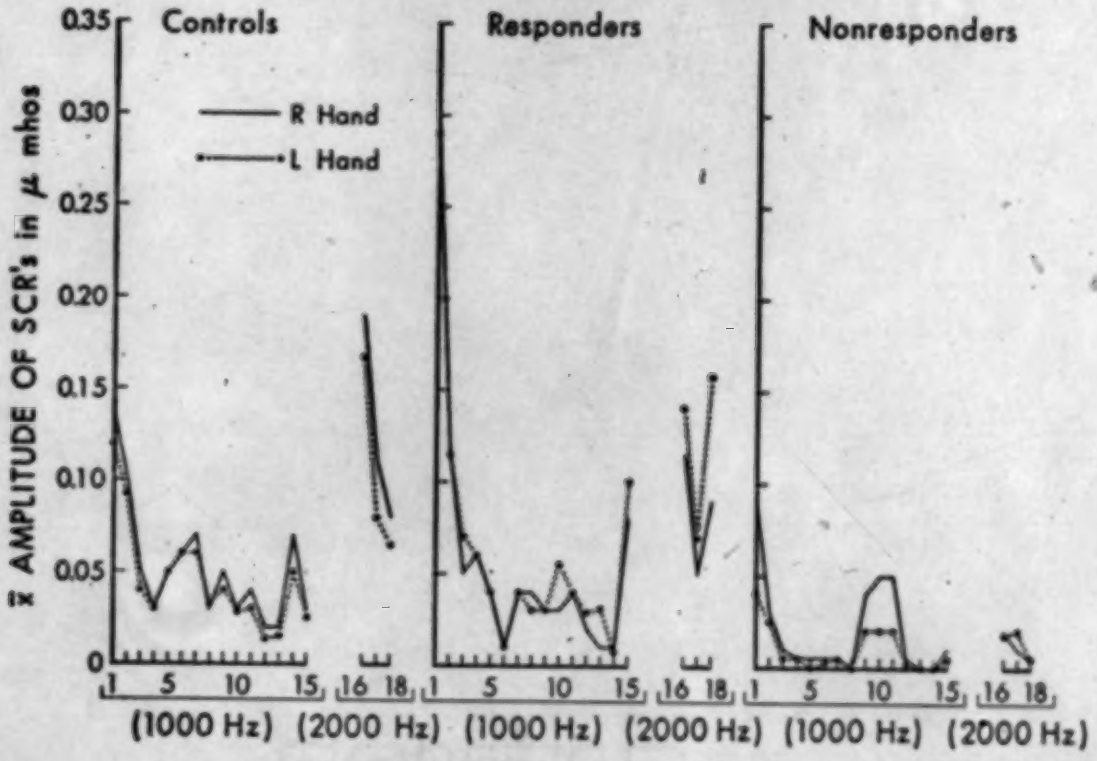


Figure 1. Mean amplitudes of SCRs for the R and L hands of the three groups during habituation (1000 Hz) and dishabituation (2000 Hz) of task two.

TABLE E

Means for normals (NC), Responders (R), and Nonresponders (NC) on SCR amplitude in Figure 1 and Scheffe test of significance ($p < .05$). Any two means not underscored by the same line are significantly different. Any two means underscored by the same line are not significantly different.

1.	R	NC	NR	R	NC	NR
	.31	.14	.09	.30	.12	.04
2.	R	NC	NR	R	NC	NR
	.13	.10	.03	.12	.09	.03
3.	R	NC	NR	R	NC	NR
	.05	.05	.01	.07	.04	.006
4.	R	NC	NR	R	NC	NR
	.06	.03	.006	.06	.03	.005
5.	NC	R	NR	NC	R	NR
	.05	.04	.005	.05	.04	.001
6.	NC	R	NR	NC	R	NR
	.06	.01	.005	.06	.01	.004
7.	NC	R	NR	NC	R	NR
	.07	.04	.005	.06	.05	.005
8.	R	NC	NR	R	NC	NR
	.04	.03	.00	.03	.03	.00
9.	NC	NR	R	NC	R	NR
	.05	.04	.03	.04	.03	.02
10.	NR	NC	R	R	NC	NR
	.05	.03	.03	.05	.03	.02
11.	NR	NC	R	R	NC	NR
	.05	.04	.04	.04	.03	.02
12.	R	NC	NR	R	NC	NR
	.02	.02	.005	.03	.01	.003
13.	NC	R	NR	R	NC	NR
	.02	.01	.00	.03	.02	.00
14.	NC	R	NR	NC	R	NR
	.07	.01	.00	.05	.009	.00

TABLE E (continued)

	RIGHT HAND			LEFT HAND		
15.	R	NC	NR	R	NC	NR
	.08	.03	.01	.10	.03	.004
16.	NC	R	NR	NC	R	NR
	.19	.13	.02	.17	.15	.02
17.	NC	R	NR	NC	R	NR
	.11	.05	.01	.08	.07	.02
18.	R	NC	NR	R	NC	NR
	.09	.08	.005	.16	.07	.005

TABLE F

Mean amplitude correlated t probabilities between 18 trials in Figure i for normal controls.

	Hand	2	3	4	5	6	7	8	9
1.	Right	.163	.004	.003	.007	.049	.023	.003	.006
	Left	.284	.013	.004	.045	.069	.046	.002	.005
2.			.099	.051	.140	.210	.235	.003	.106
			.168	.084	.234	.239	.269	.004	.050
3.				.259	1.000	.549	.268	.351	.955
				.314	.560	.479	.357	.533	.768
4.					.418	.136	.055	.854	.483
					.333	.115	.070	.975	.602
5.						.550	.314	.386	.938
						.665	.598	.421	.525
6.							.828	.021	.527
							.973	.038	.168
7.								.017	.344
								.057	.124
8.									.291
									.483

TABLE F (continued)

	Hand	10	11	12	13	14	15	16	17	18
1.	Right	.001	.002	.001	.001	.039	.004	.366	.312	.172
	Left	.002	.010	.001	.002	.046	.005	.394	.160	.078
		.022	.071	.014	.008	.515	.042	.058	.622	.555
		.024	.097	.030	.025	.334	.078	.044	.813	.194
		.351	.106	.041	.030	.543	.199	.011	.045	.404
		.412	.064	.069	.059	.641	.292	.024	.122	.488
		.947	.800	.145	.121	.361	.675	.011	.037	.226
		1.000	.939	.088	.275	.371	.773	.023	.040	.229
		.338	.042	.060	.061	.416	.298	.015	.056	.436
		.310	.017	.087	.066	.884	.270	.025	.209	.620
		.060	.247	.008	.005	.894	.084	.028	.166	.578
		.020	.173	.008	.013	.784	.080	.042	.271	.758
		.012	.076	.003	.001	.959	.050	.020	.149	.655
		.031	.101	.006	.005	.734	.065	.036	.274	.791
		.731	.734	.460	.317	.347	.925	.004	.015	.110
		.950	.980	.387	.470	.469	.857	.007	.005	.070
		.133	.443	.088	.084	.482	.367	.013	.039	.419
		.273	.629	.099	.107	.523	.483	.015	.022	.265
			.856	.189	.146	.265	.745	.007	.014	.196
			.947	.177	.251	.349	.843	.013	.008	.121
				.144	.148	.233	.582	.011	.028	.283
				.288	.296	.138	.793	.017	.065	.282
					.793	.143	.331	.008	.014	.101
					.605	.113	.154	.015	.012	.078
						.161	.321	.005	.013	.079
						.127	.455	.011	.013	.086
							.300	.064	.216	.782
							.300	.048	.275	.695
								.011	.026	.169
								.024	.036	.182
									.054	.060
									.054	.014
										.425
										.042

TABLE G

Mean correlated t probabilities between 18 trials in Figure 1 for responders.

	Hand	2	3	4	5	6	7	8	9
1.	Right	.161	.072	.078	.057	.044	.060	.061	.053
	Left	.051	.018	.016	.010	.007	.010	.012	.010
2.			.051	.307	.075	.015	.028	.038	.024
			.098	.331	.051	.004	.008	.008	.006
3.				.878	.667	.022	.360	.111	.027
				.886	.453	.026	.315	.020	.017
4.					.431	.321	.587	.603	.613
					.406	.305	.743	.582	.600
5.						.299	.885	.910	.857
						.309	.774	.804	.835
6.							.082	.089	.066
							.051	.182	.103
7.								1.000	.857
								.446	.398
8.									.797
									.818

TABLE G (continued)

	Hand	10	11	12	13	14	15	16	17	18
1.	Right	.055	.057	.050	.046	.043	.141	.182	.075	.151
	Left	.012	.010	.010	.005	.007	.136	.023	.013	.214
2.		.029	.034	.026	.026	.014	.417	.925	.081	.574
		.049	.013	.024	.069	.004	.831	.612	.096	.781
3.		.315	.088	.025	.114	.017	.603	.145	.880	.588
		.642	.085	.011	.313	.022	.602	.180	.943	.485
4.		.555	.577	.395	.355	.321	.866	.332	.841	.739
		.894	.680	.549	.621	.306	.699	.267	.907	.480
5.		.711	.861	.489	.364	.297	.598	.085	.709	.447
		.729	.916	.773	.877	.308	.498	.090	.470	.384
6.		.376	.063	.596	.903	.356	.263	.031	.106	.197
		.237	.025	.284	.498	.356	.260	.033	.071	.266
7.		.542	.908	.364	.177	.076	.508	.057	.371	.386
		.749	.754	.466	.623	.047	.500	.041	.241	.368
8.		.716	.604	.202	.297	.083	.410	.082	.381	.408
		.524	.268	.851	.972	.152	.293	.070	.155	.351
9.		.827	.884	.219	.277	.043	.395	.054	.360	.379
		.500	.281	.763	1.000	.075	.328	.053	.112	.339
10.			.769	.658	.416	.337	.459	.028	.094	.330
			.729	.550	.458	.220	.589	.014	.448	.389
11.				.191	.288	.060	.409	.071	.368	.391
				.356	.776	.025	.408	.071	.246	.372
12.					.827	.518	.236	.063	.231	.240
					.901	.247	.283	.071	.169	.309
13.						.805	.333	.029	.147	.139
						.469	.468	.015	.234	.214
14.							.252	.029	.093	.190
							.252	.031	.063	.261
15.								.540	.630	.906
								.699	.654	.744
16.									.103	.569
									.063	.907
17.										.521
										.440

TABLE H - Nonresponders

Correlated t probabilities unavailable. Computer program DER 5: ANOV12 unable to run due to a lack of variance.

APPENDIX B

Summary of Analyses of Electrodermal Latency

TABLE A

Correlated t-test probabilities between the 7 latency conditions of Figure 5 for normal subjects.

	Hand	2	3	4	5	6	7
1.	Right	.513	.954	.043	.701	.092	.000
	Left	.542	.240	.039	.187	.034	.000
2.			.574	.263	.681	.204	.006
			.879	.259	.921	.118	.034
3.				.024	.395	.065	.000
				.103	.823	.141	.000
4.					.042	.969	.000
					.079	.632	.000
5.						.061	.000
						.057	.000
6.							.000
							.000

TABLE B

Correlated t-test probabilities between the 7 latency conditions of Figure 5 for responding schizophrenics.

	Hand	2	3	4	5	6	7
1.	Right	.577	.978	.402	.343	.029	.978
	Left	.346	.881	.362	.202	.032	.939
2.			.109	.767	.470	.062	.270
			.127	.761	.815	.148	.038
3.				.289	.397	.040	.632
				.235	.266	.052	.519
4.					.754	.127	.564
					.603	.141	.462
5.						.038	.399
						.021	.344
6.							.067
							.084

TABLE C

Correlated t-test probabilities between the 7 latency conditions of Figure 5 for nonresponding schizophrenics.

	Hand	2	3	4	5	6	7
1.	Right	.218	.107	.246	.691	.535	.089
	Left	.549	.157	.093	.604	.569	.110
2.			.571	.634	----	----	.025
			.969	.629	----	----	.079
3.				.925	.097	.181	.661
				.911	.041	.190	.504
4.					.672	.389	.060
					.659	.449	.330
5.						.217	.497
						.225	.156
6.							.219
							.218

Bilateral Asymmetry of Latency Scores

Response latencies were investigated for bilateral asymmetries since it was noted during testing that some schizophrenics had large differences (but without any notable consistency). Latency asymmetries were investigated during physical stimulation (task 1), startle, (task 8), and shock trials (task 7) since these conditions were the only ones producing enough SCRs in the nonresponder group to make analysis feasible. Latency difference scores between the right and left hand were computed for each response available. The mean absolute value and average value of the differences are presented in Table D for all three groups. The mean average value of the latency differences did not differ between the three groups. The mean absolute value of the latency differences was greater for the nonresponding group only. Hence, the nonresponders had a greater degree of SCR asymmetry, compared with controls (t (23) = 2.17, $p < .05$, 1-tailed) and responders (t (13) = 2.04, $p < .05$, 1-tailed). Bull and Gale (1975) investigated latency differences in normals during tone stimulation and reported a mean right-left latency difference of .20 seconds in their normal subjects.

TABLE D

Latency Difference

Mean of right hand minus left hand latency differences for conditions 1, 2, 3, and 7 of Figure 5.

Groups	N	Absolute Value of the difference	Average Value of the difference
Normals	17	$\overline{X} = .12$ $s^2 = .0102$	$\overline{X} = -.08$
Responding Schizophrenics	7	$\overline{X} = .13$ $s^2 = .0057$	$\overline{X} = -.07$
Nonresponding Schizophrenics	8	$\overline{X} = .32$ $s^2 = .0626$	$\overline{X} = -.07$

One-tailed t-tests of absolute value of the difference

Nonresponders/normals	Nonresponders/responders
$t(23) = 2.17$	$t(13) = 2.04$
$p < .05$	$p < .05$

APPENDIX C

Summary of Ascent Time and Ratios Analyses

TABLE A

Correlated t-test probabilities between the thirteen situations in Figure 6 of ascent times and ratios for normal controls.

Ascent Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.191	.885	.000	.000	.001	.000	.000	.401	.000	.000	----	.000
	Left	.306	.665	.000	.000	.000	.000	.000	.879	.000	.000	.000	.000
2.			.152	.000	.000	.003	.000	.000	.475	.000	.000	----	.002
3.			.170	.000	.000	.001	.000	.000	.304	.000	.000	.000	.005
				.000	.000	.000	.000	.000	.562	.000	.000	----	.000
4.				.000	.000	.000	.000	.000	.580	.000	.000	.000	.000
					.164	.080	.497	.016	.000	.002	.856	----	.056
5.					.077	.158	.401	.035	.000	.013	.923	.554	.032
						.559	.070	.523	.000	.003	.099	----	.587
6.						.542	.131	.323	.000	.008	.084	.105	.206
							.033	.860	.005	.001	.061	----	.854
7.							.095	.601	.001	.004	.067	.073	.514
								.235	.000	.006	.263	----	.566
8.								.038	.000	.008	.155	.175	.241
									.000	.000	.001	----	.895
9.									.000	.000	.000	.003	.809
										.000	.000	----	.001
10.										.000	.000	.000	.000
											.027	----	.000
11.											.039	.008	.001
												----	.041
12.												.488	.016

													.000

Ascent Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.796	.225	.002	.002	.011	.002	.006	.001	.000	.000	----	.006
	Left	.996	.862	.000	.002	.001	.003	.003	.000	.000	.000	.000	.091
2.			.676	.000	.001	.006	.001	.006	.003	.000	.000	----	.187
3.			1.000	.000	.001	.000	.005	.008	.027	.000	.000	.000	.318
				.002	.003	.011	.022	.006	.001	.000	.000	----	.010
4.				.001	.002	.001	.003	.002	.000	.000	.000	.000	.014
					.510	.748	.009	.005	.004	.056	.024	----	.008
5.					.784	.100	.006	.005	.002	.008	.009	.036	.004
						.912	.025	.017	.007	.276	.096	----	.016
6.						.419	.040	.040	.007	.074	.049	.172	.014
							.182	.052	.030	.499	.201	----	.048
7.							.284	.017	.006	.314	.111	.690	.007
								.006	.002	.262	.569	----	.018
8.								.375	.025	.775	.321	.967	.026
									.260	.006	.259	----	.064
9.									.051	.225	.998	.210	.026
										.001	.004	----	.076
10.										.001	.001	.000	.088
											.152	----	.001
11.											.052	.927	.005
												----	.002
12.												.038	.000

													.000

TABLE B

Correlated t-test probabilities between the thirteen situations in Figure 6 of ascent times and ratios for responders.

Ascent Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.208	.170	.004	.066	.004	.005	.003	.072	.004	.004	----	.013
	Left	.073	.142	.014	.093	.001	.000	.001	.023	.002	.001	.001	.017
2.			.767	.041	.340	.020	.010	.103	.646	.003	.015	----	.055
3.			.949	.126	.459	.036	.007	.081	.763	.003	.026	.009	.053
4.				.003	.272	.009	.005	.029	.866	.002	.016	----	.005
5.				.001	.208	.002	.003	.010	.683	.003	.006	.000	.003
6.					.092	.854	.885	.053	.071	.008	.192	----	.947
7.					.116	.643	.219	.893	.204	.027	.318	.033	.295
8.						.382	.219	.534	.590	.044	.144	----	.255
9.						.565	.118	.464	.662	.041	.234	.046	.181
10.							.781	.032	.017	.017	.132	----	.929
11.							.344	.101	.031	.076	.123	.021	.307
12.								.124	.009	.009	.278	----	.674
13.								.070	.011	.040	.714	.415	.435
									.163	.008	.033	----	.142
									.083	.053	.035	.024	.406
										.014	.002	----	.196
										.033	.006	.030	.201
											.253	----	.059
											.463	.533	.250
												----	.115
												.369	.661

													.251

Ascent Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.056	.423	.002	.024	.004	.043	.030	.047	.000	.000	----	.149
	Left	.321	.302	.007	.050	.015	.052	.022	.107	.013	.007	.000	.065
2.			.889	.003	.030	.013	.053	.040	.068	.001	.001	----	.312
3.			.438	.008	.047	.023	.051	.018	.112	.035	.005	.001	.119
4.				.002	.033	.005	.048	.069	.016	.001	.001	----	.080
5.				.015	.058	.053	.042	.024	.090	.046	.010	.013	.126
6.					.230	.043	.145	.028	.056	.442	.249	----	.004
7.					.645	.095	.186	.024	.719	.326	.370	.076	.024
8.						.487	.638	.119	.588	.273	.298	----	.117
9.						.331	.593	.244	.995	.473	.552	.446	.189
10.							.380	.413	.321	.033	.038	----	.503
11.							.304	.503	.214	.197	.059	.340	.571
12.								.113	.800	.903	.782	----	.091
13.								.296	.470	.996	.717	.617	.035
									.814	.032	.001	----	.320
									.374	.770	.015	.891	.059
										.193	.600	----	.039
										.742	.626	.509	.148
											.528	----	.014
											.334	.531	.786
												----	.038
												.225	.024

													.249

Correlated t-test probabilities between the thirteen situations in Figure 6 of ascent times and ratios for nonresponders.

Ascent Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.805	.362	.011	.275	.288	.920	.977	.177	----	----	----	.111
	Left	.901	.519	.057	.212	.117	.088	.445	.114	----	----	----	.292
2.			.764	.482	.333	.030	----	----	.942	----	----	----	.309
			.775	.580	----	.186	----	----	.622	----	----	----	.694
3.				.110	.139	.391	.818	.779	.875	----	----	----	.111
				.153	.467	.333	.379	.687	.863	----	----	----	.215
4.					.816	.666	.360	.446	.021	----	----	----	.360
					.656	.513	.100	.416	.019	----	----	----	.224
5.						----	.247	.342	.303	----	----	----	.945
						----	.981	.291	.253	----	----	----	.874
6.							----	----	.228	----	----	----	.884
							----	----	.157	----	----	----	.322
7.								.395	.353	----	----	----	.574
								.521	.328	----	----	----	.626
8.									.272	----	----	----	.674
									.240	----	----	----	.622
9.										----	----	----	.082
										----	----	----	.008
10.											----	----	----
											----	----	----
11.												----	----
												----	----
12.													----

Ascent Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13
1.	Right	.896	.773	.672	.906	.259	.131	.066	.174	----	----	----	.578
	Left	.290	.534	.998	.982	.215	.721	.759	.079	----	----	----	.577
2.			.283	.753	.626	.400	----	----	.192	----	----	----	.339
			.587	.923	----	.415	----	----	.438	----	----	----	.133
3.				.483	.125	.357	.992	.964	.293	----	----	----	.790
				.044	.370	.553	.659	.679	.063	----	----	----	.323
4.					.189	.813	.458	.450	.122	----	----	----	.739
					.899	.155	----	----	.103	----	----	----	.656
5.						----	----	----	.081	----	----	----	.440
						----	----	----	.969	----	----	----	.341
6.							.454	.445	.171	----	----	----	.403
							.103	.046	.156	----	----	----	.430
7.								.641	----	----	----	----	----
								.984	----	----	----	----	----
8.									----	----	----	----	----
									----	----	----	----	----
9.										----	----	----	.733
										----	----	----	.096
10.											----	----	----
											----	----	----
11.												----	----
												----	----
12.													----

TABLE D

Independent t-test comparisons between normals and responders of ascent time and ascent ratio for each of the 13 situations illustrated in Figure 6. N/A = not available.

Normals/Responders

	Right			Left			
	df	t	p	df	t	p	
1.	22	1.6	.13	22	1.6	.12	Time
	22	.67	.51	22	1.5	.14	Ratio
2.	21	1.7	.10	21	.02	.04	
	21	-.24	.81	21	.52	.61	
3.	22	3.1	.006	22	3.1	.005	
	22	.21	.83	22	-.09	.93	
4.	22	.81	.47	22	-.06	.96	
	22	.79	.44	22	.88	.39	
5.	21	-.44	.67	21	-.67	.51	
	21	.81	.43	21	.66	.52	
6.	19	1.8	.09	19	1.3	.21	
	19	1.03	.32	19	.07	.14	
7.	21	.77	.45	22	1.18	.25	
	21	-.04	.97	22	.25	.81	
8.	21	1.39	.18	21	1.43	.17	
	21	-.21	.84	21	.27	.79	
9.	22	2.3	.03	22	2.6	.01	
	22	-1.5	.15	22	-1.9	.07	
10.	19	1.26	.22	18	.50	.58	
	19	.44	.67	18	1.2	.26	
11.	21	1.11	.28	21	.90	.38	
	21	-.99	.37	21	-1.2	.23	
12.		N/A		22	1.56	.13	
		N/A		22	1.02	.32	
13.	19	1.7	.10	19	2.0	.07	
	19	-1.0	.33	19	-1.1	.30	

TABLE E

Independent t-test comparisons between normals and nonresponders of ascent time and ascent ratio for each of the thirteen situations illustrated in Figure 6. N/A = not available.

Normal/Nonresponders

	Right			Left			
	df	t	p	df	t	p	
1.	23	1.6	.12	23	.61	.54	Time
	23	-3.1	.005	23	-3.3	.003	Ratio
2.	18	.67	.51	18	.13	.26	
	18	-8.4	.0000	18	-5.9	.0000	
3.	22	.4	.68	22	.36	.73	
	22	-4.1	.0004	22	-3.2	.004	
4.	22	1.5	.14	22	.06	.12	
	22	.18	.86	21	-1.3	.22	
5.	17	-.78	.45	16	.34	.74	
	17	-.63	.52	16	-.54	.59	
6.	17	-.78	.45	17	.42	.83	
	18	-.06	.95	18	-2.6	.02	
7.	17	-.21	.84	17	.30	.77	
	17	-2.8	.01	17	-1.9	.07	
8.	16	.39	.78	16	.36	.71	
	16	-4.5	.0004	16	-3.3	.005	
9.	20	.20	.84	20	.21	.42	
	20	-9.1	.0000	20	-5.3	.0000	
10.		N/A			N/A		
		N/A			N/A		
11.		N/A			N/A		
		N/A			N/A		
12.		N/A			N/A		
		N/A			N/A		
13.	19	.001	1.00	19	.46	.93	
	19	-2.5	.02	19	-2.6	.02	

TABLE F

Independent t-test comparisons between responders and nonresponders of ascent time and ascent ratio for each of the thirteen situations illustrated in Figure 6. N/A = not available.

	Responders/Nonresponders						
	df	t	p	df	t	p	
1.	13	-.04	.97	13	-.7	.49	Time
	13	-1.9	.07	13	-2.1	.06	Ratio
2.	9	-.36	.73	9	-.24	.81	
	9	-5.7	.0003	9	-3.9	.004	
3.	12	-1.2	.27	12	-.99	.39	
	12	-2.6	.02	12	-2.0	.07	
4.	12	-1.6	.13	12	-1.1	.30	
	12	-.9	.40	11	-2.2	.05	
5.	8	.62	.55	7	.51	.62	
	8	-1.51	.17	7	-1.02	.34	
6.	8	-3.4	.010	8	-2.6	.03	
	9	-1.7	.125	9	-2.3	.05	
7.	6	-1.4	.21	7	-.80	.45	
	6	-2.6	.04	7	-2.17	.07	
8.	7	-.54	.61	7	-.57	.58	
	7	-3.9	.006	7	-4.1	.005	
9.	10	-1.9	.09	10	-2.7	.02	
	10	-4.7	.0009	10	-2.7	.02	
10.		N/A			N/A		
		N/A			N/A		
11.		N/A			N/A		
		N/A			N/A		
12.		N/A			N/A		
		N/A			N/A		
13.	10	-1.5	.18	10	-1.9	.08	
	10	-1.4	.19	10	-1.5	.17	

APPENDIX D

Summary of SCR Recovery Time and Ratio Analyses

TABLE A

Correlated t-test probabilities between the fourteen situations in Figure 7a and 7b of recovery times and ratios for the normal controls.

Recovery Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.079	.007	.006	.382	.221	.013	.005	.000	.000	.000	.000	----	.001
	Left	.117	.000	.044	.347	.173	.006	.002	.000	.000	.000	.000	.000	.001
2.			.674	.007	.423	.065	.004	.012	.000	.002	.001	.001	----	.005
			.752	.020	.908	.031	.010	.021	.001	.001	.001	.001	.001	.009
3.				.004	.353	.007	.003	.006	.001	.000	.000	.002	----	.003
				.008	.442	.005	.003	.004	.001	.000	.000	.002	.000	.002
4.					.049	.281	.412	.802	.000	.000	.000	.001	----	.015
					.083	.841	.325	.312	.000	.001	.000	.000	.000	.032
5.						.207	.028	.067	.004	.013	.006	.006	----	.026
						.086	.059	.085	.020	.017	.010	.024	.015	.048
6.							.054	.354	.000	.000	.000	.000	----	.024
							.226	.542	.000	.001	.000	.001	.001	.086
7.								.017	.000	.000	.000	.000	----	.057
								.320	.000	.000	.000	.000	.000	.097
8.									.000	.000	.000	.000	----	.006
									.000	.000	.000	.000	.000	.105
9.										.015	.010	.184	----	.000
										.001	.039	.016	.000	.000
10.											.405	.382	----	.002
											.918	.008	.676	.012
11.												.616	----	.002
												.098	.217	.001
12.													----	.001
													.123	.002
13.														----
														.000

Recovery Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.130	.004	.000	.017	.007	.020	.006	.014	.000	.027	.008	---	.230
	Left	.311	.008	.000	.019	.001	.028	.010	.149	.010	.071	.301	.005	.374
2.			.502	.008	.004	.005	.118	.427	.706	.813	.576	.325	----	.248
			.141	.000	.012	.001	.025	.028	.746	.207	.511	.238	.060	.743
3.				.027	.032	.024	.459	.727	.112	.825	.198	.137	----	.050
				.017	.026	.006	.276	.291	.243	.948	.352	.151	.468	.247
4.					.883	.461	.074	.014	.010	.001	.008	.017	----	.012
					.619	.291	.053	.005	.007	.001	.007	.010	.022	.015
5.						.440	.014	.014	.016	.042	.018	.026	----	.023
						.410	.010	.020	.017	.038	.017	.034	.031	.018
6.							.087	.035	.022	.026	.022	.030	----	.036
							.049	.014	.008	.006	.007	.006	.012	.017
7.								.187	.111	.186	.088	.080	----	.046
								.813	.063	.130	.068	.038	.346	.091
8.									.322	.460	.099	.056	----	.022
									.101	.433	.104	.075	.914	.115
9.										.507	.625	.487	----	.049
										.093	.512	.090	.196	.489
10.											.042	.241	----	.018
											.040	.002	.241	.014
11.												.941	----	.163
												.897	.004	.366
12.													----	.009
													.018	.012
13.														----
														.005

TABLE B

Correlated t-test probabilities between the fourteen situations in Figures 7a and 7b of recovery times and ratios for the responding schizophrenics.

Recovery Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.160	.802	.056	.062	.193	.072	.149	.133	.121	.067	.039	----	.089
	Left	.762	.474	.043	.022	.215	.048	.096	.102	.098	.056	.023	.038	.106
2.			.126	.119	.370	.311	.123	.263	.201	.189	.103	.070	----	.148
			.500	.109	.313	.316	.148	.232	.217	.210	.109	.066	.072	.158
3.				.010	----	.235	.010	.104	.223	.190	.091	.045	----	.001
				.004	----	.223	.025	.105	.145	.137	.087	.031	.164	.235
4.					.286	.556	.137	.081	.122	.094	.318	.040	----	.418
					.064	.544	.159	.168	.117	.113	.432	.037	.117	.565
5.						.300	.240	.234	.274	.513	.701	.066	----	.013
						.075	.195	.318	.537	.968	.074	.238	.822	.190
6.							.883	.467	.062	.232	.069	.088	----	.156
							.949	.594	.045	.168	.041	.073	.204	.177
7.								.673	.154	.139	.199	.083	----	.239
								.818	.169	.173	.269	.067	.116	.336
8.									.056	.043	.028	.009	----	.050
									.042	.048	.028	.012	.025	.110
9.										.244	.004	.845	----	.053
										.210	.849	.197	.284	.360
10.											.589	.122	----	.730
											.550	.113	.420	.700
11.												.241	----	.676
												.224	.972	.700
12.													----	.101
													.037	.064
13.														----
														.575

Recovery Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.157	.269	.012	.131	.072	.005	.060	.084	.001	.016	.140	----	.394
	Left	.175	.319	.029	.160	.152	.017	.065	.442	.004	.098	.193	.007	.335
2.			.459	.050	.187	.159	.063	.053	.328	.025	.001	.631	----	.468
			.441	.079	.192	.205	.038	.055	.798	.026	.048	.426	.318	.701
3.				.729	----	.112	.782	.538	.764	.384	.592	.867	----	.264
				.365	----	.582	.891	.271	.520	.070	.535	.889	.731	.037
4.					.440	.661	.408	.736	.217	.244	.439	.147	----	.041
					.677	.498	.483	.393	.215	.755	.357	.955	.476	.244
5.						.455	.548	.443	.638	.593	.570	.208	----	.168
						.994	.660	.197	.770	.779	.989	.250	.345	.254
6.							.807	.939	.464	.753	.561	.526	----	.091
							.972	.361	.155	.833	.797	.901	.742	.073
7.								.842	.279	.316	.720	.357	----	.093
								.546	.353	.413	.941	.635	.067	.187
8.									.636	.982	.477	.357	----	.086
									.442	.538	.296	.427	.215	.077
9.										.247	.478	.592	----	.036
										.268	.314	.433	.328	.357
10.											.456	.911	----	.005
											.724	.481	.423	.083
11.												.782	----	.003
												.933	.521	.041
12.													----	.556
													.589	.581
13.														----
														.667

TABLE C

Correlated *t*-test probabilities between the fourteen situations in Figures 7a and 7b of recovery times and ratios for the nonresponding schizophrenics.

Recovery Time

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.386	.751	.063	.428	.347	----	.568	----	----	----	----	----	.603
	Left	.371	.973	.164	.618	.483	.611	.471	----	----	----	----	----	.731
2.			.629	.334	----	.362	----	----	----	----	----	----	----	.623
			.558	.506	----	.500	----	----	----	----	----	----	----	.926
3.				.115	.269	.142	----	.070	----	----	----	----	----	.389
				.168	.114	.240	.374	.235	----	----	----	----	----	.700
4.					.747	.125	----	.306	----	----	----	----	----	.024
					.394	.869	.982	.326	----	----	----	----	----	.150
5.						----	----	.988	----	----	----	----	----	.049
						----	.604	.361	----	----	----	----	----	.578
6.							----	----	----	----	----	----	----	.331
							----	----	----	----	----	----	----	.368
7.								----	----	----	----	----	----	----
								.104	----	----	----	----	----	.685
8.									----	----	----	----	----	.458
									----	----	----	----	----	.117
9.										----	----	----	----	----
										----	----	----	----	----
10.											----	----	----	----
											----	----	----	----
11.												----	----	----
												----	----	----
12.													----	----
													----	----
13.														----

Recovery Ratio

	Hand	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	Right	.522	.492	.412	.623	.225	----	.740	----	----	----	----	----	.620
	Left	.212	.550	.540	.700	.524	.161	.971	----	----	----	----	----	.854
2.			.479	.180	.444	.503	.942	.611	----	----	----	----	----	.651
			.509	.704	----	.652	----	----	----	----	----	----	----	.732
3.				.357	.226	.694	----	.067	----	----	----	----	----	.231
				.406	.430	.434	.002	.494	----	----	----	----	----	.464
4.					.467	.732	----	.934	----	----	----	----	----	.241
					.687	.838	.075	.474	----	----	----	----	----	.785
5.						----	----	.406	----	----	----	----	----	.430
						----	.514	.385	----	----	----	----	----	.305
6.							----	----	----	----	----	----	----	.283
							----	----	----	----	----	----	----	.654
7.								----	----	----	----	----	----	----
								.167	----	----	----	----	----	.068
8.									----	----	----	----	----	.418
									----	----	----	----	----	.196
9.										----	----	----	----	----
										----	----	----	----	----
10.											----	----	----	----
											----	----	----	----
11.												----	----	----
												----	----	----
12.													----	----
													----	----
13.														----

TABLE D

Independent t-test comparisons between normals and responders of recovery time and ratio for each of fourteen situations illustrated in Figures 7a and 7b. N/A = not available.

	Right			Left			
	df	t	p	df	t	p	
1.	21	-.56	.58	21	-.51	.62	Time
	21	-.13	.90	21	.31	.76	Ratio
2.	19	.93	.36	18	.46	.65	
	19	.47	.65	18	-.39	.70	
3.	15	-.03	.97	15	-.49	.63	
	15	-1.4	.17	15	-2.4	.03	
4.	21	1.3	.21	21	.1	.20	
	21	.70	.49	21	1.3	.20	
5.	15	1.6	.14	15	1.5	.15	
	15	.70	.52	15	1.1	.29	
6.	18	.72	.48	19	.26	.80	
	18	.77	.46	19	1.2	.26	
7.	20	-.26	.79	21	-.21	.84	
	20	.05	.96	21	-.26	.80	
8.	20	-.21	.84	20	-.31	.76	
	20	-.82	.42	20	-1.3	.20	
9.	19	.30	.77	18	-.80	.44	
	19	-.10	.92	18	.27	.55	
10.	19	-.25	.80	18	-.28	.78	
	19	-.94	.36	18	-.19	.85	
11.	21	-.94	.36	21	-.9	.38	
	21	-2.0	.06	21	-1.8	.08	
12.	18	-.08	.94	19	.30	.77	
	18	-.75	.47	19	-1.3	.21	
13.	N/A			21	-.68	.50	
	N/A			21	-.05	.96	
14.	18	.66	.52	18	.92	.37	
	18	-.74	.47	18	-.62	.54	

TABLE E

Independent t-test comparisons between normals and nonresponders of recovery time and ratio for each of the fourteen situations illustrated in Figures 7a and 7b. N/A = not available.

	Right			Left			
	df	t	p	df	t	p	
1.	20	-1.1	.28	20	-1.6	.12	Time
	20	-3.2	.004	20	-3.6	.002	Ratio
2.	17	.96	.35	15	.57	.58	
	17	-3.6	.002	15	-3.4	.004	
3.	16	-1.2	.25	17	-1.5	.15	
	17	-2.6	.02	17	-2.2	.05	
4.	21	1.08	.29	20	.12	.90	
	21	-.39	.70	20	-.86	.40	
5.	13	.62	.54	13	.03	.97	
	13	-.31	.76	13	-1.0	.33	
6.	16	.56	.59	17	.01	.99	
	15	-.54	.60	17	-1.2	.25	
7.	N/A			16	-1.2	.26	
	N/A			16	-6.2	.0000	
8.	16	.21	.84	15	.95	.36	
	16	-4.7	.0003	15	-3.5	.003	
9.	N/A			N/A			
	N/A			N/A			
10.	N/A			N/A			
	N/A			N/A			
11.	N/A			N/A			
	N/A			N/A			
12.	N/A			N/A			
	N/A			N/A			
13.	N/A			N/A			
	N/A			N/A			
14.	18	-4.4	.0004	17	-4.0	.001	
	18	-4.0	.0008	17	-2.9	.01	

TABLE F

Independent t-test comparisons between responders and nonresponders of recovery time and ratio for each of the fourteen situations illustrated in Figures 7a and 7b. N/A = not available.

	Right			Left			
	df	t	p	df	t	p	
1.	11	-.32	.75	11	-.68	.51	Time
	11	-2.1	.06	11	-2.4	.04	Ratio
2.	8	.18	.86	7	.18	.86	
	8	-2.5	.04	7	-2.00	.08	
3.	5	-.75	.49	6	-.57	.59	
	6	-1.0	.35	6	.21	.43	
4.	12	-.35	.73	11	-1.1	.28	
	12	-1.2	.25	11	-3.0	.01	
5.	4	-1.1	.33	4	-2.9	.05	
	4	-1.09	.33	4	-2.5	.06	
6.	8	-.12	.91	8	-.19	.86	
	7	-1.3	.24	8	-2.1	.07	
7.	N/A			7	-.29	.78	
	N/A			7	-6.6	.0003	
8.	6	.21	.84	7	.73	.49	
	6	-2.5	.04	7	-1.	.36	
9.	N/A			N/A			
	N/A			N/A			
10.	N/A			N/A			
	N/A			N/A			
11.	N/A			N/A			
	N/A			N/A			
12.	N/A			N/A			
	N/A			N/A			
13.	N/A			N/A			
	N/A			N/A			
14.	10	-3.2	.01	9	-5.5	.0004	
	10	-2.5	.03	9	-1.7	.12	

APPENDIX E

Bilateral Skin Conductance Level Differences

Gruzelier and Venables (1973) reported higher SC levels of the right hand in their responder group and higher SC levels of the left hand in their nonresponder group and no consistent differences in SC level for the control group. They interpreted the higher amplitude of SCRs in the right hand of both schizophrenic groups, regardless of asymmetry of SC levels, and reversed SC level asymmetries for psychopathological groups with comparable high SC level values, responders having higher right hand levels and depressives having higher left hand levels, as supportive of Flor-Henry's (1969) theory of laterality of dysfunction corresponding with type of psychopathy (Venables, 1975). As Venables himself states, caution is needed due to lack of sufficient evidence for ipsi- versus contra- lateral psychophysiological effects of CNS disturbances (Venables, 1975).

Of interest then in the present study was any evidence of bilateral asymmetry of SCR amplitudes or SC levels. As reported previously, no consistent bilateral SCR amplitude differences were found. With respect to SC level differences, the averages of right hand minus left hand SCL change scores did not reveal any consistent right or left hand differences in any of the three experimental groups. In fact, the trends observed were in the opposite direction: higher SC levels of the left hand in the responding group and higher SC levels in the right hand for the nonresponding group, compared to those reported by Gruzelier and Venables (1973).

In order to determine if the degree of SCl asymmetry differed significantly between groups, the absolute value of the difference in right hand and left hand levels (side of highest SCL difference ignored) was analyzed. Figure iia (appendix E) illustrates the mean of the absolute value of the difference scores for each group across several tasks in the session. The greatest degree of SCL difference occurred in the control group followed by the nonresponder and responder schizophrenic groups, respectively. A two way repeated measures analysis of variance on the SCL absolute value of the difference scores was used to test for the group and situations differences shown in Figure iia (appendix E). The three groups did not differ significantly; however, the repeated measures effect was highly significant (F (5,145) = 4.6, $p < .001$).

The absolute SCL difference scores of each subject were then transformed by a simple transformation used in previous data collection at AHE in order to allow a comparison with data collected in this laboratory on other normal controls and subjects with known unilateral brain dysfunction. This transformation consists of dividing each subjects' SCL difference by the sum of the right and left hand values and adjusts for the fact that bilateral variations of subjects with low SCL values would be less than those for subjects with high SCL values. In this laboratory we have observed that the index score for normals appears to vary between .0 and a rare extreme of .27 whereas some unilaterally brain damaged subjects had index scores as high as .50. In examining the individual index scores of all subjects in the present study it was found that the maximum score in the control group

was .24 and in the responding group .23. In contrast, 4 of the 8 nonresponders had scores between .28 and .33. In three of these cases the right hand level was higher than the left hand level; the reverse occurred in the fourth.

Utilization of SCL asymmetries as one index of brain damage (see Parsons, 1970) in the present study supports presence of brain damage in 4 out of 8 nonresponders and none of the control group or responding subjects. Figure iib (appendix E) presents the average of the index scores across the same repeated measures as in Figure iia. It can be seen that the controls and the responders do not change their relative position with respect to each other whereas the non-responders do. A two-way repeated measures analysis of variance on the index scores approached significance for the groups main effect ($p < .07$) and the groups by repeated measures effect ($p < .09$) and was significant for the repeated measures effect, F (5,145) = 4.8, $p < .001$.

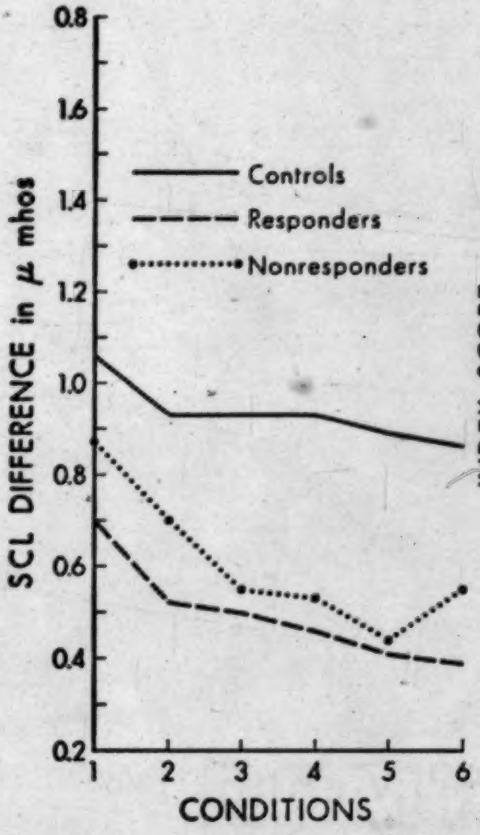


Figure iia

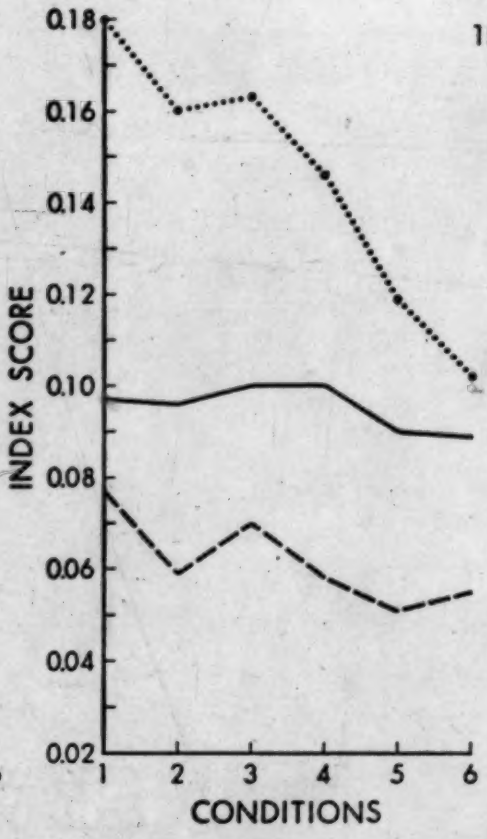


Figure iib

Figure iia & b. (a) The absolute SCL differences between R and L hands and (b) the Index scores of absolute SCL differences for the three groups during the six conditions below. For each condition the SCL's were those measured in the latency period of the SCRs scored during these tasks. (1) task one, physical stimulation, (2) task two, habituation/dishabituation task, (3) neutral tones, and (4) signal tones of task three, (5) cognitive tasks four and five, and (6) task seven, shock, and task eight, startle.

APPENDIX F

Summary of WAIS and Neuropsychological Variables

TABLE A

Means for (a) Normals, (b) Responders and (c) Nonresponders on neuropsychological variables and Scheffe test of significance ($p < .05$). Any two means not underscored by the same line are significantly different. Any two means underscored by the same line are not significantly different.

		A Normals		B Responders		C Nonresponders	
	Variable	N		N		N	
1.	Wepman-Jones - Errors	13	1.46	7	11.29	6	11.0
2.	Speech Sounds - Errors	13	4.46	7	12.57	6	13.0
3.	Trail Making A + B	13	85.77	7	182.14	6	253.17
4.	Trail Making A	13	25.69	7	42.14	6	47.17
5.	Trail Making B	13	60.38	7	140.00	6	206.00
6.	Memory for Designs	13	0.77	7	4.7	6	9.17
7.	Colored Progressive Matrices	13	1.08	7	3.86	6	9.67
8.	Finger Tapping-Preferred Hand	13	50.08	7	56.00	6	57.83
9.	Finger Tapping-Non-Preferred Hand	13	53.69	7	59.57	6	61.17
10.	Symbol Gestalt	13	2.62	6	3.20	6	3.34
11.	Halstead Category	13	27.54	6	55.00	6	115.17
12.	Finger Localization-Preferred Hand	13	3.00	7	4.43	6	9.83
13.	Finger Localization-Non-Preferred Hand	13	3.69	7	6.14	6	13.00
14.	Organic Integrity Test	13	21.46	7	48.00	6	34.67
15.	Minute Estimation-Mean	13	63.08	6	35.83	6	26.83
16.	Minute Estimation-Deviation from 60	13	20.54	6	24.17	6	33.17

TABLE A (continued)

	Variable	A Normals N		B Responders N		C Nonresponders N	
17.	Tactual Performance - Preferred	13	262.77	7	644.29	6	720.00
18.	Tactual Performance - Non-Preferred	13	212.46	7	363.00	5	742.40
19.	Tactual Performance - Both	13	134.00	7	292.43	5	382.40
20.	Tactual Performance - Localization	13	4.85	7	7.00	6	8.17
21.	Tactual Performance - Memory	13	2.69	7	2.86	6	5.67
22.	Tactual Performance - Total	13	608.46	7	1299.71	6	1657.33
23.	Seashore Rhythm	13	1.92	7	6.14	6	12.00
24.	Retinal Rivalry - Mean	13	21.31	6	16.50	6	15.00
25.	Retinal Rivalry - Deviation from 13.14	13	11.73	6	4.69	6	6.33
26.	Oral Word Fluency	13	31.62	7	40.43	6	40.17
27.	Perdue Pegboard - Preferred Hand	13	10.31	7	11.43	6	14.67
28.	Perdue Pegboard - Non-Preferred Hand	13	10.31	7	11.14	6	13.33
29.	Perdue Pegboard - Both	13	12.31	7	13.71	6	16.67
30.	Perdue Pegboard - Assemblies	13	42.23	7	48.43	6	61.83
31.	Perdue Pegboard - Total	13	76.38	7	74.29	6	51.33
32.	Face-Hands	13	0.08	6	2.33	6	4.33

TABLE B

WAIS

Group	Score: Verbal	Performance	Total
Responding Schizophrenics	90.7	93.0	91.7
Nonresponding Schizophrenics	80.2	73.0	76.0

APPENDIX G

Medication effects in schizophrenics

In the present study, as well as in many related studies, the psychophysiological effects observed in the schizophrenics are confounded with the effects of medication on the autonomic nervous system. In the present study, an attempt was made to deal with this problem by analyzing respiration rhythms which may not be affected by the major tranquilizers and by assessing the relationship between the medication level and the SC level and number of spontaneous fluctuations which are known to be attenuated by tranquilizers. Furthermore Gruzelier's recent work (personal communication, 1975) was mentioned which provided evidence that the responder and nonresponder schizophrenics did not change their category after being removed from medication for four weeks. Skin conductance levels however, did increase and the SCL bilateral asymmetry observed under medication was enhanced when subjects were unmedicated. The responder and nonresponder schizophrenic groups in the present study both contained one schizophrenic who was unmedicated and others whose medication was minimal.

From these observations, it was suggested that the psychophyiological effects observed could not be attributed solely to medication effects. However, the possibility for complex interactive effects between response to medication and type of schizophrenics was not ruled out. Furthermore, Gruzelier's data on unmedicated schizophrenics did not deal with the medication effects on other electrodermal measures such as latency and recovery time. Therefore, the results of the present study have to be considered as tentative until they have been resplicated with unmedicated schizophrenics.

APPENDIX H

Univariate versus Multivariate Statistics

It could be argued that the statistics of choice in the present study should have been a multivariate analysis. A multivariate technique such as Hotelling's T^2 test would have controlled for the large number of variables considered in the study (see A primer of multivariate statistics, by R. J. Harris, Academic press, 1975).

However, the application of multivariate analysis in the present study could not be done because data was missing for many of the variables under different conditions. The major reason for the missing data was due to the intrinsic nature of the data. That is, under several conditions or tasks where no changes in amplitude occurred there would be no meaningful data for latency, ascent time, and recovery time. Since this phenomena occurred in no predictable fashion for the many stimulus presentations and occurred often enough in all three groups, sufficient data was not available for a proper multivariate analysis.